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The Effects of Sleep Deprivation on Information Processing, New Learning, and Memory in Mildly Head-Injured Subjects With Post-Concussion Symptoms.

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THE EFFECTS OF SLEEP DEPRIVATION ON INFORMATION PROCESSING, NEW LEARNING, AND MEMORY IN MILDLY HEAD-INJURED SUBJECTS WITH POSTCONCUSSION SYMPTOMS

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ABSTRACT

Subjects who had sustained a mild head injury (MHI) and uninjured control subjects were examined before and after 36 hours of sleep deprivation. MHI subjects and uninjured controls were selected from among individuals who scored above the 50th percentile on the Postconcussion Syndrome Checklist (PCSC), a measure designed to assess the frequency, intensity and duration of postconcussion symptoms. Sixty subjects were divided into four groups: Head injured/sleep deprived, head injured/non-sleep deprived, uninjured/sleep deprived, and uninjured/non-sleep deprived. Performance was compared among the groups on the Selective Reminding Test (SRT) and the Paced Auditory Serial Addition Test (PASAT), which are designed to measure memory and new learning, and information processing, respectively. The SRT and the PASAT were administered to all subjects three times: Learning trial, pre-sleep deprivation trial, and post-sleep deprivation trial. Performance did not differ between groups on any aspect of the SRT or PASAT post-sleep deprivation. A significant trials effect was found, in which performance declined across administrations for all groups on the Delayed Recognition and Delayed Recall aspects of the SRT prior to sleep deprivation. The performance of all groups steadily improved across trials on the PASAT prior to sleep deprivation. There was no effect of sleep deprivation on the SRT or PASAT performances. Results indicated that MHI subjects can maintain a level of performance commensurate with that of uninjured controls following 36 hours of sleep deprivation.

A number of factors may have prevented relations between head injury, sleep deprivation, and performance from emerging: (1) MHI subjects may not have sustained an injury severe enough to result in diffuse brain injury or produce neuropsychological deficits; (2) MHI and uninjured subjects endorsed PCS symptoms at a level that was one and one-half standard deviations lower than would be considered clinically significant for PCS; (3) MHI subjects may not have experienced PCS symptoms immediately following injury, thereby decreasing the likelihood that PCS symptoms endorsed during the current study were injury-related; (4) the duration of sleep deprivation may not have been long enough to elicit performance deficits on neuropsychological measures; and (5) the neuro-psychological measures administered may not have been of sufficient duration to elicit performance decrements following sleep deprivation.

REVIEW OF THE LITERATURE

The literature review will provide an overview of the incidence of head injury, and will describe the factors which place one at risk for sustaining a head injury and influence the rate and level of recovery. Means of defining mild head injury (MHI) will be delineated. Concussion and diffuse axonal injury (DAI), both of which commonly result from head injury, will be discussed. The pattern of neuropsychological deficits that occurs following head injury will be described, with particular attention paid to changes in information processing, memory and learning. Postconcussion syndrome will be discussed, as will its relation to the persistence of post-injury cognitive deficit. Finally, the effects of sleep deprivation on cognitive functions in general, and following MHI, will be described.

Incidence

The incidence of head injury in the United States is estimated to be 200/100,000 per year. This rate, applied to the 1990 United States population of 250 million, indicates that there are about 500,000 cases of head injury per year, including those who died before reaching a hospital (Kraus, 1993), as well as those evaluated at or admitted to a hospital for treatment. MHI accounts for approximately 80 percent of all head injury admissions in the United States (Levin, 1993; Marshall, 1989), with estimates ranging from 72 percent to 95 percent (Vollmer & Dacey, 1991). Most data on head injury are collected through hospital records, yet it has been estimated that 20 to 40 percent of all patients with mild head injuries in the United States do not seek

medical care (Evans, 1992; Templer et al., 1992). Thus, the overall incidence of MHI is difficult to determine, although the actual level is probably higher than estimates based solely on hospital admission data.

The incidence of head injury is highest in young people. A peak incidence occurs in adolescents and young adults aged 15-24. Secondary incidence peaks include: infants and children (Cooper, Tabaddor, & Hauser, 1983; Whitman, Cloony-Hoganson, & Desai, 1984), and the elderly (Annegers, Grabow, Kurland, 1980; Cooper et al., 1983). Males are more commonly injured than females by a ratio of 2:1 (Evans, 1992). The distribution of cases by age depends on the nature of the population studied and the external causes dominant in that population. For example, in most studies, motor vehicle accidents (MVAs) account for the major proportion of head injuries and involve a disproportionately large number of young persons (Kraus,1993), whereas falls are more likely the cause of injury among the elderly (Evans, 1992). In industrialized countries such as the United States, estimates of the relative cause of head trauma are as follows: MVA, 45%; falls, 30%; occupational accidents, 10%; recreational accidents, 10%; and assaults, 5% (Jennett & Frankowsky, 1990).

Head Injury: Causes and Effects

Impairment of function and damage to neural structures can result either from focal or diffuse head injury. Focal head injury is caused by the impact that occurs when an object hits the head, or the head hits an object. Focal injuries result in localized damage, such as laceration of the scalp, fracture of the skull,

extradural hematoma, and cerebral contusion. As focal injuries do not result in shearing of neural tissue, neurological deficits typically remit once the hematoma and/or contusion resolves.

Diffuse brain injury is the principal result of head injury (Gennarelli, Thibault, & Adams, 1985; Gennarelli et al.,1982; Strich, 1956). It is caused by shearing or tensile strains that occur when neuronal structures move relative to one another, and can occur even without impact to the cranium. The following categories of diffuse brain injury are now recognized: (1) Mild concussion: Diffuse brain injury which involves temporary disturbance of neurological function without loss of consciousness. (2) Classical cerebral concussion: A temporary, reversible neurological deficiency caused by trauma that results in loss of consciousness for less than six hours. (3) Diffuse axonal injury (DIA): Prolonged traumatic coma lasting more than six hours (Gennarelli, 1993).

Diffuse brain injuries are acceleration/deceleration injuries, the severity of which is determined by the direction, magnitude, and speed with which the head moves, from rest or to rest, during the injury sequence (Gennarelli et al., 1982). Up to a point, the amount of head acceleration or deceleration is the most important factor in determining how much brain deformation and resulting axonal damage occurs. Injury becomes more severe as head acceleration increases. However, the direction of head movements becomes critically important in more severe injuries. Experimentally, severe diffuse injury occurs only in coronal (lateral) head motion (Gennarelli et al., 1982). At equivalent or even higher accelerations, sagittal or horizontal head movements

produce diffuse injury of only mild, or at worst, moderate type (Gennarelli, 1993).

The violent head motions are themselves sufficient to produce strains and distortions within the brain, resulting in shearing or stretching of nerve fibers with consequent axonal damage. At low velocity, injurious levels of shear/strain do not extend deeper than the cortex. However, following severe trauma, damage extends inward to affect the diencephalic-mesencephalic core, including the corpus callosum, dorsolateral quadrant of the brain stem, and the deep white matter of the cerebral hemispheres (Gennarelli et al., 1982; Mandel, 1992; Ommaya & Gennarelli, 1974; Strich, 1956). Damage in this region typically results in coma and permanent neuropsychological deficits.

The severity and location of cortical and subcortical disconnections and the degree of associated structural damage depends on the material and structural properties of neuronal tissue and cerebral vascular anatomy (Ommaya, 1968). Along with axonal damage, vascular damage occurs at the capillary level and results in small, isolated or multiple hemorrhages at the gray-white matter junctions. The presence of intraventricular blood and generalized brain swelling are considered suggestive signs of DAI (Levi, Guilburd, Lemberger, Soustiel, & Feinsod, 1990). The extent of damage is also influenced by the physical properties of the skull, with its bony protrusions and dural partitions. Those parts of the cortex covered by smooth surfaces (e.g., the occipital lobes) should suffer the least damage, whereas those portions covered by rough surfaces (e.g., orbitofrontal and anterior temporal lobes)

sould suffer the most (Adams, Graham, & Scott, 1980; Ommaya & Gennarelli, 1974).

Axonal change is a consistent feature of MHI (Adams et al., 1982; Blumbergs et al., 1989; Levi et al., 1990). MHI may disrupt axons without physically shearing or tearing them (Povlishock, Becker, Cheng, & Vaughn, 1983). Jane et al. (1985) found numerous degenerating axons in the inferior colliculus, dorsolateral mesencephalic tegmentum, and basis pontis in monkeys subjected to mild acceleration/deceleration injury. Povlishock et al. (1983), however, reported that damage to a limited number of axons within a given system may not compromise the entire system's functioning. Thus, axonal changes may occur without clinically observable signs. Povlishock et al. (1983) demonstrated primary axonal changes in felines that are capable of making an uneventful recovery from minor head trauma. Axonal change appeared to be the result of a focal and discrete alteration within the axon which becomes progressively more severe and ultimately results in axonal separation.

Radiological studies have found neuronal damage following MHI. Brain stem auditory evoked responses have been found to be delayed, even in patients with very mild injuries who are free from other neurological abnormalities (Newcombe, Rabbitt, & Briggs, 1993, Shapiro & Sacchetti, 1993). Zimmerman, Bilaniuk, and Gennarelli (1978) found positive computerized tomography (CT) findings in approximately 3% to 5% in patients with mild head injury. Further, magnetic resonance imaging (MRI) studies on patients with

mild and moderate head injury revealed intracranial abnormalities (hyperintensities). Extraparenchymal (extradural) lesions at baseline were larger in patients who had moderate impairment of consciousness compared to patients with mild impairment. The groups did not differ in size of parenchymal (subdural) lesions which were distributed mainly in the frontotemporal region, a pattern consistent with neuropathological findings in fatal head injuries (Levin, Williams, Eisenberg, High, & Guinto, 1991).

Concussion

Cerebral concussion is a common result of head injury. It is defined as a clinical syndrome characterized by immediate and transient impairment of neural function, such as alteration of consciousness and disturbance of vision and equilibrium, due to mechanical forces (Caveness, 1966; Ommaya & Gennarelli, 1974). Concussion is a complex phenomenon, the nature and severity of which are determined by multiple factors.

Concussion involves the brain stem and other loci in the brain that function together to maintain wakefulness (Jane, Steward, & Gennarelli, 1985; Walker, 1973). The awake state is mediated by a complex interaction involving numerous brain stem (ascending reticular activating system (ARAS)) centers, subcortical structures including the hypothalamus, and the cerebral cortex. The disconnection of one of these structures from the others results in an altered state of consciousness. In general, the awake state requires the ARAS of the brain stem to be automatically/functionally connected to the cerebral cortex of both hemispheres. This projection may be either direct or indirect via

hypothalamic-diencephalic centers. Similarly, feedback from the cerebral cortex of both hemispheres to both the diencephalon and the reticular activating system of the brain stem is necessary for consciousness. Unconsciousness can result either from disconnection of the cortex from the diencephalon and brain stem, or by dysfunction of both cerebral hemispheres. In MHI, this dysfunction is primarily physiological and not structural. Normal brain function is sustained by a complicated balance of electrochemical events occurring in billions of cells simultaneously. Following mild injury, these events can be disrupted without causing marked structural damage to neural tissue. Thus, when the electrochemical milieu of the brain returns to normal, the usual interaction between the cerebral hemispheres and brain stem is re-established, and consciousness returns. As the severity of injury increases, structures within the brain can become physically or anatomically damaged, resulting in permanent disruption (Gennarelli, 1993).

Head trauma that does not result in a loss of consciousness (LOC) still can cause significant intracranial trauma and result in concussion (Strauss & Savitsky, 1934). Concussion that results in a temporary disturbance of neurological function without LOC is classified as mild (Gennarelli, Thibault, & Adams, 1982). Thus, persons who have sustained a MHI and are momentarily disoriented and confused, as well as patients with perceptible amnesia, can be diagnosed as having sustained a mild concussion (Binder, 1986).

Neuropsychological Deficits

After head trauma, a broad range of neuropsychological deficits may occur, with higher-level functions more vulnerable to disruption than lower-level functions. Improvement following losses occurs in complex as well as in simple functions. The initial degree of deficit plays a large role in determining the subsequent degree of recovery: those with substantial losses show a greater amount of improvement but also a greater amount of residual deficit, and those with less initial impairment show a smaller amount of improvement and a smaller remaining residual deficit (Dikmen et al., 1986).

While physical, sensory, and intelligence quotient (IQ) difficulties may well recover within a few weeks to a few months, language, memory, and attention difficulties may persist for six months or longer. Difficulties may persist in information processing abilities which might include learning under complex and stressful situations, functioning efficiently in other than routine situations, and a tendency to be more easily overwhelmed. Not only are information processing difficulties common in patients following mild brain impairment, but they tend to persist in the absence of all other measurable difficulties and may go on for several years. Patients with the aforementioned deficits can usually function quite normally in most situations, but have difficulty in situations that require efficient adaptation to changing task requirements. Maintenance of an optimal level of functioning tends to require more energy, more purposeful effort, and sometimes the utilization of strategies not required prior to the injury (Boll, 1983).

Cognitive recovery rates vary from study to study, and are influenced by the stringency of inclusion and exclusion criteria adhered to by investigators. Previous studies have suggested that substantial recovery occurs during the first year post-injury, with the majority of improvement occurring during the first three to six months (Dikmen, Reitan, & Temkin, 1983; Jennett, Snoek, & Bond, 1981; Levin et al., 1987). It is important to note that reversibility of cognitive deficits after MHI in no way excludes the presence of microscopic or otherwise subtle brain lesions (Jane et al., 1985; Oppenheimer, 1968; Povlishock et al., 1983) that may result in the development of postconcussion syndrome and/or compromised neuropsychological functioning under stress.

Information Processing

Deficits in information processing, including attention and reaction time, are among the most salient effects of MHI (Boll, 1983; Gentilini et al., 1985; Gronwall, 1989; Gronwall & Wrightson, 1981; Stuss et al., 1985; Gronwall, 1977; Gronwall & Wrightson, 1974). The pathophysiology of information processing deficits is difficult to determine. Diffuse white matter lesions (Adams et al., 1982; Ommaya & Gennarelli, 1974) and brain stem dysfunction (Barth et al., 1983; Gronwall & Sampson, 1974; Oppenheimer, 1968) may underlie impairment, as these would adversely affect processing speed and arousal level, respectively. A disruption in frontal-limbic-reticular activating system brainstem control is presented as an alternative hypothesis, as frontal, temporal, and limbic areas are particularly sensitive to head injury (Posner & Peterson, 1990; Stuss et al., 1985).

MHI adversely affects information processing capacity, which in turn affects attention and memory (Gronwall & Wrightson,1981). Concussed patients can process a limited number of items as swiftly as normal controls. However, as the number of items increases, the performance of the concussed patient declines and diverges further from that of controls. Thus, a critical point is reached when the concussed patient's channel capacity is exceeded (Beers, Goldstein, & Katz, 1994; Gronwall & Wrightson, 1974). Recovery in processing speed to a level approximating that of uninjured control subjects has been found to occur within one month post-injury (Gronwall and Wrightson, 1974 &1975; Levin et al., 1987). However, recovery from such deficits have been noted from three months (Hugenholtz, Stuss, Stethem, & Richard, 1988) to three years post-injury (Ewing, McCarthy, Gronwall, & Wrightson, 1980).

Research indicates that among head injured subjects, information processing deficits appear to be more a function of decreased processing speed than processing ability (Arcia & Gualtieri, 1994). This premise is supported by the work of Stuss et al. (1985) who administered the Stroop Color Word Test to head injured subjects. Results did not indicate a selective focused attention deficit, which would be demonstrated by decreased performance time or increased errors in the Stroop interference subtest alone. Rather, there was a general tendency for slowness in response on all Stroop subtests. Impaired performance of head injured subjects on the Trail Making Test and the Wechsler Adult Intelligence Scale (WAIS) Digit Symbol subtest also suggests decreased speed of information processing (Stuss et al., 1985).

Finally, results from complex reaction time studies indicate that head injured subjects have a divided attention deficit, defined as slowness in consciously controlled information processing (Conboy, Barth, & Boll, 1986; Levin, 1989). Thus, the aforementioned findings suggest that head injured subjects are unable to process multiple bits of information rapidly and easily.

Memory and Learning

Memory is the cognitive domain most susceptible to impairment following head injury (Levin, Goldstein, High, & Eisenberg, 1988; Levin et al., 1983). This is likely due to the high concentration of injury-related parenchymal and extraparenchymal lesions in the anterior temporal lobes (Levin et al., 1987). The anterior temporal lobes contain the hippocampus and other neuronal structures strongly implicated in the storage and retrieval of new memories (Levin, 1993).

It appears that head injury seems to have at least three different effects on memory. The first, a deficit in information processing ability, is related to performance on memory tasks only when the tasks require complex processing, or where time constraints are imposed. The second is a deficit in the ability to place material into long-term memory storage (Shapiro & Sacchetti, 1993). The third is a deficit in the ability to retrieve newly learned material from memory once it has been stored. This retrieval deficit occurs in about one quarter of closed head injury cases regardless of injury severity (Gronwall & Wrightson, 1981). Long-term recall of prior knowledge remains largely intact (Shapiro & Sacchetti, 1993).

Hall and Bornstein (1991) examined the memory of subjects who had sustained either a mild (73%) or moderate (27%) head injury. Subjects' performance on the Wechsler Memory Scale-Revised (WMS-R) Logical Memory test was examined six months post-injury. Both head-injured and control subjects showed serial position effects, yet serial position effects were different between groups: On immediate recall, both groups showed strong primacy and recency effects, but closed head injury patients recalled fewer items, particularly from the middle third of the story.

Investigations that have included follow-up examinations indicate that memory recovers over a period of one to three months after a single, uncomplicated mild head injury (Levin, 1989). There is evidence that multiple trauma potentiates the early memory disturbance following relatively mild head injury (Dikmen et al., 1986; Gentilini et al., 1985; Levin, 1989), thereby lengthening the recovery process in patients who have sustained more than one head injury.

Definition /Classification of Mild Head Injury

In the past, MHI was not clearly defined. Lack of a clear definition resulted in heterogeneous samples in which the more complicated injuries of many subjects were classified as mild. Leniency of classification has resulted in discrepant findings in the MHI literature. Several researchers have found persistent impairment of cognition 3 to 12 months after MHI (Barth et al., 1983; Rimel et al., 1981; Rutherford et al., 1979). However, others have demonstrated recovery of cognitive abilities within 3 months after MHI to a level

comparable to that of a matched control group (Gentillini et al., 1985; Gronwall & Wrightson, 1980; Levin, Mattis, Ruff et al. 1987). Adoption of uniform inclusion criteria in studies of MHI may help to reconcile such discrepant findings concerning outcome.

Several classification schemes have been proposed for the grading of head injury severity (Becker, Miller, & Greenberg, 1982; Teasdale & Jennett, 1974). The Glasgow Coma Scale (GCS) (Teasdale & Jennett, 1974) is the most widely used of such grading systems (Vollmer & Dacey, 1991). The GCS is designed to assess one's level of alertness following head injury by measuring one's ability to respond visually, verbally, and motorically to stimuli. A score of 13-15 indicates that one is aware, conversant, and able to obey motor commands. A score of 9-12 indicates that one is awake and able to localize painful stimuli, yet disoriented, less able to converse intelligibly, and less able to follow motor commands. Scores of 8 or less indicate that one is minimally responsive (e.g., unable to follow commands; opens eyes in response to painful stimuli only; flexion or withdrawal of limb to painful stimuli only) or is comatose. Since the introduction of the GCS, a series of studies have adopted the sum score of 13-15 to characterize MHI (Dikmen, McLean, & Temkin,1986; Levin et al., 1987; Rimel, Giordani, Barth, & Jane, 1982), a sum score of 9-12 to define moderate head injury, and a sum score of 8 or less to define severe head injury (Rimel et al., 1982).

The use of clinical indicants of severity has several advantages. First, the determinations are simple, prospective, reproducible, and can be

accomplished with little intraobserver or interobserver variability. Second, clinical measures can be repeated throughout the patient's course of evaluation and treatment (Vollmer & Dacey, 1991). However, there are several disadvantages of basing severity classification on the initial level of alertness at the time of injury. Some patients present with altered consciousness due to the ingestion of drugs or alcohol or to the effects of systemic injuries (Galbraith, Murray, & Patel, 1976), thereby confounding a severity rating that is intended to be based on degree of impairment from head trauma alone. Additionally, the patient who is unresponsive immediately after injury may rapidly improve to a normal neurologic state, making initial severity ratings invalid. Finally, grading systems based on levels of consciousness are insensitive to the transient neurologic derangements that accompany milder injuries where a relatively rapid return to alertness is the rule (Vollmer & Dacey, 1991). The timing of classification, therefore, is an important variable that must be considered.

In contrast to basing the classification of MHI solely on GCS score (e.g., Gentilini et al., 1985; Rimel et al., 1981; Ruff, Levin, & Marshall, 1986), investigators have begun to use additional criteria, such as length of unconsciousness, usually specified as no greater than 15-20 minutes (Levin & Amparo, 1987; Rimel & Giordani,1981), and length of hospitalization, typically 48 hours or less, to exclude more severe cases with medical complications, and/or those requiring surgical procedures and general anesthesia (Levin et al., 1987; Rimel et al., 1981). Advocates of the more restrictive diagnostic criteria cite extreme heterogeneity in pathophysiological features among

patients who have GCS scores ranging from 13 to 15 and argue that mild head injuries complicated by the presence of radiological abnormalities (e.g., focal brain lesion, depressed skull fracture, etc.) are more severe than injuries that are uncomplicated (Williams, Levin, & Eisenberg, 1990). The presence of brain stem signs (e.g., decerebrate or decorticate posturing) and/or residual motor abnormalities (e.g., apraxia, ataxia, hemiplegia) is also indicative of more severe neurological involvement (Clifton, Levin, Michel, & Choi, 1992; Gennarelli, 1993). These claims are supported by research conducted by Williams et al. (1990) who found that performance of mildly head-injured patients (normal CT scan, and either a normal skull X-ray or an abnormality limited to a linear or basilar skull fracture) surpassed that of patients with complicated mild head injuries (initial and lowest GCS of 13 to 15 and radiographic evidence of focal brain lesion, depressed skull fracture, or both) and moderate injuries (initial and lowest GCS of 9 to 12 with or without positive radiological findings) on measures of verbal fluency, information processing, and recognition memory. These results provide support for classifying patients with mild head injuries complicated by an acute radiological abnormality into a separate category.

Another method of determining the severity of brain injury is the assessment of the duration of Post Traumatic Amnesia (PTA). PTA is defined as the time between the injury and the point at which the patient has regained anterograde memory. The duration of PTA roughly correlates with injury severity (Jennett & Teasdale, 1981). Durations ranging from less than 60

minutes (Gronwall & Wrightson, 1974) to less than 24 hours (MacFlynn, Montgomery, Fenton, & Rutherford, 1984) have been used to define MHI.

The validity of PTA length as a predictor of outcome after MHI is questionable. When of short duration, PTA is difficult to assess, and is unreliably reported by patients after 3 months (Gronwall & Wrightson, 1980). Even for the severely injured, PTA only predicts approximately 25% of the variance of performance on cognitive tests (Levin, Benton, & Grossman, 1982). Its reliability is reduced if it is assumed that a patient with a clear sensorium is no longer in a state of PTA, as the patient may not become amnesic until a few hours after the trauma or may drift in and out of PTA (Binder, 1986; Gronwall & Wrightson, 1980). Finally, PTA is not a consistently valid predictor of symptomatology and disability following MHI, whether outcome is examined in terms of time off work (Steadman & Graham, 1970; Wrightson & Gronwall, 1981), persistence of postconcussion symptoms (Rutherford et al., 1979), or the degree and duration of neuropsychological impairment (Barth et al., 1983; Gronwall & Wrightson, 1974).

The above factors are essential to the formulation of a sound definition of MHI. However, additional factors that influence post-injury outcome must be considered prior to conducting research in the area of MHI. Pre-injury medical and social history and other individual differences contribute immensely to the quality of outcome after head injury. Important historical factors which must be considered include age, education, socioeconomic status, alcohol use, prior injury, and prior psychiatric or neurologic disorder. Age has been found to

influence recovery post-head injury, with older patients tending to recover more slowly and less completely than their younger counterparts (Barth et al., 1983; Rutherford, Merrett, & McDonald, 1979; Wrightson & Gronwall, 1981). For example, Barth et al. (1983) found that as age increased and education decreased, neurocognitive deficits presenting as residual sequelae increased as well.

Education bears a strong relation to outcome after head injury. Finlayson, Johnson, and Reitan (1977) state that the measures influenced by level of education have auditory-verbal and language requirements in common. It may be the case that these abilities become more highly developed, or over-learned, as level of education increases. Thus, following a head injury of equal severity, a well-educated person is likely to fare better, cognitively, than a less well-educated counterpart, as he or she has a larger cognitive reserve from which to draw. Although education is positively related to post-injury outcome, the adverse effects on cognition are often more noticeable in an individual who was functioning at a high premorbid cognitive level. Conversely, as level of education decreases, the deleterious effects of head injury on cognition are more difficult to detect, as it becomes more difficult to determine what represents a decline in functioning (Finlayson et al., 1977).

Alcohol intoxication has been reported in 35 to 42 percent of patients evaluated for MHI (Rimel, Giordani, Barth, Boll, & Jane, 1981; Rutherford, Merrett, & McDonald, 1977). There are three aspects of alcohol use that are related to brain injuries. First, alcohol is a precipitating factor in motor vehicle

accidents, falls, assaults, suicide, and recreation-related injuries (Kraus, 1993). Second, alcohol may affect the accuracy of diagnosis following head injury. Jagger, Fife, and Vernberg (1984) have noted that alcoholic intoxication at the time of diagnosis hampers assessment of severity of brain injury as it results in a lower, and therefore, more severe Glasgow Coma Scale score. Thus, an accurate clinical assessment is sometimes not possible as formal assessment procedures overestimate the severity of brain injury in patients who are intoxicated at the time of their injury and emergency room evaluation. Third, alcohol confounds the prediction of outcome after brain injury, as regular, heavy alcohol consumption results in persisting cognitive deficits and central nervous system dysfunction (Parks et al., 1991), which obscure injury-related deficits.

Previous head injury has been found to increase the time course of recovery from both mild and moderate head injuries. Theoretically, one's cognitive and neurologic reserve is compromised following head injury, and one's reserve is further diminished with each subsequent injury. Cumulative axonal damage and contusions may explain why prior head injury is a risk factor for slower, and potentially less complete recovery from a more recent head injury (Gronwall & Wrightson, 1975).

It appears that the best means of defining MHI is to use a clear, restrictive definition, and to limit the use of classification measures that provide ratings subject to miscalculation following MHI, e.g., the GCS and estimates of PTA (Binder, 1986; Gronwall & Wrightson, 1980; Vollmer & Dacey, 1991).

Thus, criteria for defining MHI should include the following: (1) Any alteration of mental status at the time of the accident, e.g., feeling dazed, disoriented, or confused (Kay et al., 1993); (2) LOC of no greater than 15 to 20 minutes (Levin & Amparo, 1987; Rimel & Giordani, 1981); (3) Length of hospitalization of 48 hours or less (Levin et al., 1987); and (4) Absence of radiological abnormalities, e.g., focal brain lesion, depressed skull fracture (Williams et al., 1990). For purposes of research, it is important to include subjects with only one head injury (Gronwall & Wrightson, 1975), to control for level of education (Finlayson et al., 1977), and to eliminate subjects with a history of alcohol abuse (Parks et al., 1991) and individuals who were intoxicated at the time of evaluation, as these variables represent potential confounds of cognitive performance.

Postconcussion Syndrome

Post concussion syndrome (PCS) is a cluster of symptoms that includes memory difficulty, headache, vertigo, depression, anxiety, concentration difficulty, blurred vision, fatigue, irritability, and photophobia. Although the symptoms typically associated with PCS are generally agreed upon, the etiology of PCS is controversial. Whereas some investigators believe the primary cause of PCS is cerebral dysfunction (Binder, 1986; Rutherford et al., 1977), others believe that PCS may initially have an organic basis, but persists because of psychological factors (Levin, 1982). Variation in the onset and course of PCS symptoms makes it difficult to unequivocally support one position. In some cases, early symptoms (within one day of injury) occur and

persist for months or years (Lindvall, Linderoth, & Norlin, 1974; Wrightson & Gronwall, 1980), but in other cases, the onset of symptoms is delayed for several weeks after injury. Late onset symptoms are considered indicative of a psychogenic etiology (Lindvall, 1974; Rutherford et al., 1979). However, the inability to rule out definite clinico-pathologic correlations makes this a risky inference, as damage following diffuse MHI is microscopic and therefore not visible on MRI or CT scans. Thus, without an autopsy, it is impossible to support or refute the role of neuropathology in late-onset PCS symptoms.

The fact that PCS symptoms have been found to develop along different time lines lends credence to the premise that PCS results from both organic and psychogenic causes. Initial PCS symptoms such as headache, dizziness, nausea are most likely organic in nature whereas late-onset symptoms that may have an organic basis (e.g., memory and concentration difficulties) may become increasingly evident as one attempts to resume his or her premorbid level of functioning (Alves, Colohan, O'Leary, Rimel, & Jane, 1986; Rutherford et al., 1979; Wood, Novack, & Long, 1984). The victim of mild to moderate head trauma may find on attempts to resume activities, that premorbid efficiency and output are difficult to attain due to subtle memory difficulties, poor concentration, and diminished speed of cognitive processing (Novack & Long, 1984). The injured person may then respond with increased effort, resulting in rapid fatigue, which further decreases efficiency and compounds deficient cognitive functioning (Gronwall & Wrightson, 1974; Van Zomeren & Van Den Burg, 1985). Fatigue and stress involved in attempting to overcome

cognitive deficits may exacerbate existing somatic problems, such as headache and dizziness (Rimel el al., 1981).

Symptoms associated with PCS occur, albeit with less frequency and intensity, in people who have not sustained a head injury (Caveness, 1966; Dikmen et al., 1986; Gouvier, Uddo-Crane, & Brown, 1988; Levin, 1989). Caveness (1966) was the first to document a prevailing level of PCS symptoms in uninjured subjects by comparing the complaints of Korean War veterans who sustained head injuries of varying severity to an uninjured group of military personnel matched for age and service in Korea. Dickmen and colleagues (1986) also found that symptoms typically reported after mild head injury were endorsed, though less frequently, by uninjured subjects. Whereas headaches, irritability, and anxiety were the symptoms reported most frequently by control subjects (Caveness, 1966; Dickmen et al., 1986), excessive fatigability, problems with memory, and inability to concentrate were reported least often (Caveness, 1966). Thus, the presence of PCS symptoms after mild head injury must be considered in relation to their prevailing level in a general population (Levin, 1989) in order to determine if one is endorsing symptoms at an abnormally high frequency and intensity, or of an abnormally long duration. It may be that, following head injury, individuals pay increased attention to cognitive and somatic difficulties believed to be injury-related. Concern over the etiology and effects of PCS symptoms may lead to anxiety and depression, which further diminishes efficiency in daily functioning (Bohnen et al., 1992; Long & Novack, 1986).

The psychogenic aspect of PCS may result, in part, from limited information provided to patients regarding the course of recovery following head injury. While many mild head injuries are managed without medical attention, those that are productive of symptoms (dizziness, nausea, vomiting, sleep at abnormal times of day, vertigo) and/or brief periods of unconsciousness, commonly come to medical attention. Medical treatment most typically is observation, physical examination, occasionally electroencephalographic examination and, in instances of more significant concussion with longer periods of unconsciousness (5 to 30 minutes), hospitalization for up to 48 hours. The findings of all of these examinations are commonly negative either immediately post-injury or shortly thereafter. In such instances, the patient is frequently assured that no neurological damage has been done. Thus, by implication, the patient is led to believe that no neurological basis exists for his or her current or future cognitive deficits (Boll,1983). Without the provision of information regarding possible post-injury sequelae such as fatigue, trouble concentrating, etc., prognosis, and management (e.g., initially maintain a light schedule), a patient may be more prone to develop PCS. Support for this conceptualization is as follows: One sustains a MHI which he or she believes to be uncomplicated; he or she immediately engages in a pre-injury level of activity and begins to experience the sequelae associated with PCS (e.g., memory and concentration difficulties, headache, fatigue) (Gronwall & Wrightson, 1979); he or she does not attribute these symptoms to his or her injury and strives to overcome them, resulting in

increased fatigue and symptom exacerbation (Gronwall & Wrightson, 1974; Van Zomeren & Van Den Burg, 1985).

Endorsement of PCS symptoms appears to be a covariate of neuropsychological impairment. Research indicates that MHI patients who report postconcussion symptoms often have measurable neuropsychological deficits, the severity of which appear to be independent of the neurological status observed immediately following injury (Leininger, Gramling, Farrell, Kreutzer, & Peck, 1990). Gronwall and Wrightson (1974) report that lower scores on the Paced Auditory Serial Addition Test (PASAT) are associated with the presence of PCS symptoms. Further, Bohnen et al. (1992) found that subjects who continued to report PCS symptoms six months after having sustained a MHI, exhibited neuropsychological deficits, specifically in the areas of selective attention, divided attention, and information processing.

Sleep Deprivation

Sleep deprivation adversely affects attentional, psychomotor, and memory performance, as well as mood and motivation (Browne et al., 1994; Jaques, Lynch, & Samkoff, 1990; Lingenfelser et al., 1994). Neuropsychological performance and mood status have been found to decline significantly following 24 hours of sleep deprivation (Bonnet, 1994; Lingenfelser et al., 1994). Further, medical students have been found to perform significantly worse on written exams following 24 hours of sleep deprivation (Jacques et al., 1990). However, one need not be completely deprived of sleep for detrimental cognitive and emotional effects to occur.

Shortening one's sleep period to a sub-optimal level (four hours or less of uninterrupted sleep) may produce similar effects (Browne et al., 1994).

A number of theories have been advanced suggesting a relationship between information processing, new learning, and sleep. One line of thought has maintained that the general capacity to receive and process information is fatigued during the day's activities and is restored during rapid eye movement (REM) sleep (Herscovitch, Stuss, & Broughton, 1980). Others have proposed that the sleep process is essential to memory consolidation and transfer of memories into long-term storage (Herscovitch et al., 1980). It is thus plausible that sleep deprivation results in decreased ability to learn new information (memory consolidation) and impaired information processing.

The relationship between cognitive functioning and sleep has been demonstrated in studies investigating the effects of various sleep rations on normal populations. Sleep deprived individuals frequently experience difficulties in sustained mental operations and have periods of misperception and disorientation (Friedman, Bigger, & Kornfield, 1971). Further, stress associated with sleep loss adversely affects all properties of information processing systems (Broadbent, 1971). Sleep deprived subjects make attempts to narrow their range of attention in a manner similar to methods of coping with cognitive overload under other conditions of stress (Hockey, 1970), but increased fatigue and decreased vigilance impede attempts to remain focused on relevant stimuli.

Sleep-deprived subjects are less able to adequately perceive and register newly presented material. Perceptual ability is likely compromised by brief lapses in consciousness due to brief intrusions of sleep, or "microsleeps", resulting in an inability to perceive new information and rehearse old items while additional items are being presented (Bonnet, 1994). Even when perceived correctly, it appears that the strength of the resulting memory traces are reduced under conditions of sleep loss. Retention is further decreased when subjects are required to "hold" items in memory for a short interval. Taken together, these findings indicate that sleep loss reduces one's ability to attend, especially for extended periods of time (Elkin & Murray, 1974). Erratic attention decreases the opportunity for information to be integrated into short-term, and subsequently long-term memory storage. Thus, sleep deprivation has been found to impede new learning. Immediate recall scores have been found to deteriorate significantly after one night of sleep loss (Gieseking, Williams, & Lubin, 1957; Nilsson, Backman, & Karlson, 1989).

In order for tasks to be sensitive to sleep deprivation, their duration must be at least 30 minutes, and preferably one hour (Jacques, Lynch, & Samkoff, 1990; Donnell, 1969). Individuals undergoing sleep loss can usually rally momentarily to perform at non-sleep-deprived levels, but the ability to maintain that performance becomes increasingly limited as task duration and sleep deprivation progress (Bonnet, 1994). Tasks of long duration interact with sleep deprivation to produce greater decrements in performance (Johnson, 1982). This is consistent with findings discussed in the information processing section:

One can override fatigue and maintain an optimal level of concentration for a brief period under conditions of stress and/or information overload, but tasks of long duration erode one's cognitive reserve, eventually resulting in compromised vigilance, information processing, and new learning ability.

The above findings have real-world implications. College students frequently deprive themselves of sleep for purposes of studying, socializing, or both. As the college semester progresses and scholastic demands increase, students report a continuous decline of time allotted for sleep (Hawkins & Shaw, 1992). Because sleep deprivation adversely affects information processing and new learning, the ability to memorize relevant material and ignore extraneous information in preparation for an exam is compromised. Students who have sustained a head injury may be especially prone to the debilitating effects of sleep deprivation, as information processing and attentional abilities may already be compromised due to brain injury. Thus mild cognitive dysfunction may be exacerbated by sleep deprivation, even to the extent that typically unmeasurable dysfunction becomes evident.

Summary and Hypotheses

Research has shown that following a MHI, persons are often not able to perform cognitive functions at a premorbid level of efficiency (Gronwall & Wrightson, 1974 & 1975; Levin et al., 1987; Long & Novack, 1986). Memory (Levin, Goldstein, High, & Eisenberg, 1988; Levin et al., 1983) and information processing (Boll, 1983; Gentilini et al., 1985; Gronwall, 1989; Gronwall & Wrightson, 1981; Stuss, Ely, Hugenholtz, Richard, LaRochelle, Poirier, et al.,

1985; Gronwall, 1977; Gronwall & Wrightson, 1974) are particularly susceptible to deficits following MHI. Further, head-injured subjects who endorse postconcussion symptoms are more likely to develop neuropsychological deficits than non-reporters (Bohnen et al., 1992; Gronwall & Wrightson, 1974; Leininger et al., 1990). Finally, sleep deprivation has been found to impede information processing, new learning, and memory (Bonnet, 1994; Elkin & Murray, 1974; Gieseking, Williams, & Lubin, 1957; Nilsson, Backman, & Karlson, 1989). Therefore, an interaction between mild head injury with PCS and sleep deprivation was hypothesized, such that the effects of the two variables are additive with respect to performance on neuropsychological tests.

Prior to sleep deprivation, head-injured and uninjured control subjects who report symptoms of PCS were expected to perform similarly on measures of memory, new learning, and information processing. Performance was expected to be similar between groups prior to sleep deprivation, as the cognitive functioning of mildly head-injured and uninjured persons is similar in the absence of stress (Ewing, McCarthy, Gronwall, & Wrightson, 1980), and when rate of stimuli presentation is kept at a constant, moderate pace (Beers, Goldstein, & Katz, 1994; Gronwall & Wrightson, 1974). Following sleep deprivation, cognitive performance was expected to deteriorate for both head-injured and control subjects, as sleep deprivation has been found to impede information processing, new learning, and memory (Bonnet, 1994; Elkin & Murray, 1974; Gieseking, Williams, & Lubin, 1957; Nilsson, Backman, & Karlson, 1989). However, the performance of head-injured PCS subjects was

expected to be significantly more impaired than that of uninjured control PCS subjects across all aspects of memory, new learning, and information processing measures. Therefore, greater impairment of performance following sleep deprivation was expected among head-injured subjects, as lack of sleep was expected to potentiate the subtle injury-related cognitive deficits that exist among head-injured subjects (Gronwall & Wrightson, 1974 & 1975; Levin et al., 1987; Long & Novack, 1986).

METHOD

Subjects and Subject Selection

Subjects were recruited from among the undergraduate population enrolled in psychology courses at Louisiana State University. Prior to involvement in the study, subjects were asked to read and sign an informed consent statement (Appendix A). A total of 159 potential subjects underwent preliminary screening which consisted of completing the Head Injury Epidemiology Questionnaire (HIEQ) (Ryan et al., in press) (Appendix B), the Postconcussion Syndrome Checklist (PCSC) (Gouvier, Cubic, Jones, Brantley, & Cutlip, 1992) (Appendix C), the Peabody Picture Vocabulary Test (PPVT) (Dunn & Dunn, 1981) (Appendix D), the Beck Depression Inventory (BDI) (Beck, 1978) (Appendix E), the State-Trait Anxiety Inventory (STAI) (Speilberger, 1983) (Appendix F), and the Structured Clinical Interview for the Diagnostic and Statistical Manual Third Edition-Revised (SCID) (APA, 1994). Head-injured and uninjured control subjects who did not have a potentially confounding neurologic (e.g., seizure disorder, attention deficit disorder) or psychiatric condition (e.g., mood disorder, anxiety disorder, psychosis, substance abuse), who were not taking medication that may interfere with and/or alter cognition (e.g. antidepressant, anxiolytic, antipsychotic, anticonvulsant, sedative hypnotic medications), and, in the case of head-injured subjects, who met the previously specified criteria for mild head injury (Kay et al., 1993) were asked to participate in the study. Additionally, it was proposed that MHI and uninjured subjects would only be included in the study if

they obtained a PCSC total score that was one and one-half standard deviations higher than that obtained by MHI and uninjured subjects without PCS in a study conducted by Gouvier et al. (1992). In the Gouvier et al. (1992) study, MHI and uninjured subjects without PCS obtained PCSC total scores of 71.35 and 64.85, respectively. Thus, MHI and uninjured subjects would have had to obtain respective PCSC total scores of 87.33 and 78.76 to meet proposed PCSC criteria for inclusion in the present study. Sixty-five suubjects were screened during the preliminary phase of the present study, however, only two subjects met the proposed PCSC criteria for inclusion. Thus, a dissertation committee-based decision was made to perform a median split on PCSC data obtained from the 65 subjects during the preliminary screening. The median PCSC total score was found to be 57.0 for MHI subjects and 56.0 for uninjured subjects. Subjects who obtained a PCSC total score that was above the 50th percentile were included in the study. This same inclusion criteria was applied in subsequent screening sessions. The mean PCSC total score for subjects included in the present study was 68.16 and 64.90 for MHI and uninjured subjects, respectively.

The screening session and experiment took place on two separate occasions. Thus, subjects who met participatory criteria were provided with an appointment time at which to return to complete the study. Upon their return, subjects were assigned to one of four groups: (1) sleep deprived head injured subjects with postconcussion symptoms, (2) sleep deprived uninjured subjects with postconcussion symptoms, (3) non-sleep deprived head injured subjects

with postconcussion symptoms, (4) non-sleep deprived uninjured subjects with postconcussion symptoms. During the course of screening, 75 subjects met previously defined exclusion criteria and were eliminated from further participation: Forty-six subjects were disqualified as they did not score above the 50th percentile on the PCSC. Eleven subjects were disqualified due to medication and/or a confounding neurological condition. Nineteen subjects were disqualified as their scores on the BDI, STAI, or both fell in the clinically significant range. An additional 15 subjects did not keep their appointment to participate in the study post-screening. Finally, nine subjects stated that they no longer wished to participate in the experiment and were released, with credit, during the experimental protocol. It should be noted that all subjects who requested to discontinue participation were released prior to the collection of baseline data and thus, prior to sleep deprivation. The final sample consisted of a total of 60 subjects; four groups, each with 15 subjects. Groups did not differ on the basis of age, race, sex, education, PCSC score, and analog intelligence quotient (I.Q.) derived from the PPVT-R standard score.

Selection Materials

The HIEQ and the PCSC were used as screening instruments. The HIEQ is a self-report instrument designed to assess for head injury and potentially confounding factors (e.g., neurological disorders and medications). If prospective subjects had ever sustained a head injury, they were asked to provide additional injury-related information, including how and when the injury occurred, and whether the injury resulted in medical examination and/or

hospitalization. Additionally, respondents were asked to provide information regarding the severity of their injury as gauged by several factors, including the amount of time that they were unconscious, length of hospitalization, and radiological findings.

Individuals who met the established criteria for MHI (Kay et al., 1993) were invited to complete the next aspect of screening. According to the literature, one was classified as mildly head injured if length of unconsciousness did not exceed 20 minutes, the injury was not complicated by the presence of radiological abnormalities (e.g., focal brain lesion, depressed skull fracture, etc.), and there was no history of a prior neurological or psychiatric disorder (Levin & Amparo, 1987; Levin, Mattis, 1987; Williams et al., 1990).

Although the Glasgow Coma Scale (GCS) is frequently used to determine the severity of a head injury, use of this measure was not possible in the present study, as subjects' medical records were not available. For many subjects, medical records did not exist, as a substantial number of mild head injuries were not medically evaluated. The inability to include the GCS among the criteria for MHI was not expected to compromise diagnostic capability, as with the exception of providing a numerical indicant of MHI, GCS scores in the mild range provide little additional information regarding the injury. Previous research has found that GCS scores in the mild range are prone to error, as they may underestimate the severity of injury that is complicated by a lesion, hematoma, or skull fracture (Williams et al., 1990). Conversely, moderate GCS

ratings may overestimate the severity of injury in a patient who is initially unresponsive, but rapidly improves to a normal neurologic state (Vollmer & Dacey, 1991). The criteria mentioned in the previous paragraph thus are appropriate and sufficiently reliable to define MHI in the absence of information provided by the GCS.

Head injured and uninjured subjects next completed the PCSC. The PCSC is a self-report measure designed to assess the frequency, intensity, and duration of nine core postconcussion symptoms, as experienced over the previous two months. Symptoms rated by the PCSC include headaches, dizziness, irritability, memory problems, difficulty concentrating, visual disturbance, aggravation by noise, judgment problems, and anxiety (Binder, 1986; Gouvier et al., 1992). Respondents rate the frequency, intensity, and duration of each symptom on a five-point Likert scale. Four symptom scores can be derived from the PCSC: a frequency total, an intensity total, a duration total, and a total score across the three dimensions. Each of the four derived scores has been shown to correlate significantly with the more established, yet less concise, Postconcussion Checklist (Gouvier et al., 1992; Oddy, Humphrey, & Uttley, 1978). The authors report that the PCSC total score best differentiates between persons with and without PCS symptoms. As previously mentioned, a median split was conducted on PCSC total score data obtained from 65 potential subjects during the preliminary screening session. The median PCSC total score was determined separately for two groups of pilot subjects (34 head injured, 31 uninjured). Head injured and uninjured subjects

who scored at or above the respective group median were asked to continue on through the screening process. The group medians were found to be 56.0 and 57.0 for uninjured subjects and head injured subjects, respectively.

Of the psychological sequelae associated with MHI and PCS, depression is most commonly reported, followed by anxiety (Bohnen et al., 1992; Klonoff, Campbell, & Klonoff et al., 1993). Depression and anxiety disrupt concentration, which diminishes efficiency in daily functioning (American Psychiatric Association, 1994; Long & Novack, 1986). Anxiety and depression represent potential confounds in the present study, as decreased concentration associated with anxiety and mood disorders may adversely affect performance on neuropsychological tests. Thus, subjects were administered the BDI and the STAI during initial screening procedures, in order to rule out clinically significant depression (BDI score $\geq$ 15) and anxiety (STAI State t-score $\geq$ 65; STAI Trait t-score $\geq$ 65).

The BDI is the most frequently used self-report method for assessing level of depression. It has been shown to have adequate internal consistency (Beck & Steer, 1987), with alpha coefficients of .86 and .81 for psychiatric and non-psychiatric populations, respectively. The BDI has also been shown to have concurrent validity with clinician ratings of depression (Brumberry, Oliver, & McClure, 1978). Subjects with a BDI score of 15 or higher were excluded from further participation, as this level has been reported to be indicative of clinically significant levels of depressive symptoms (Beck & Steer, 1987).

The STAI is a brief, self-report measure of state and trait anxiety which has been shown to have adequate reliability and validity. Alpha coefficients for the state and trait anxiety components of the instrument range from .90 to .92 (Speilberger, 1983). Individuals with a state or trait t-score of 65 or greater were excluded from further participation as this level is reported to be indicative of clinically significant anxiety (Speilberger, 1983).

Subjects were also interviewed to rule out the presence of diagnosable psychiatric disorders (e.g., major depression, dysthymia, bipolar disorder, anxiety disorder, psychosis, and substance abuse) which may affect performance on neuropsychological tests. The Structured Clinical Interview for the Diagnostic and Statistical Mannual of Mental Disorders Third Edition - Revised (SCID) was used to conduct the screening interview. The purpose of the screening was not formal diagnosis, but to identify and exclude subjects who met diagnostic criteria for major psychiatric disorders. Thus, subjects were excluded if they reported significant psychiatric symptoms, even if sufficient information was not present to formulate a definite diagnosis. Mild personality disorders were not a basis for exclusion.

Education and intelligence quotient (IQ) have been found to significantly influence performance on some neuropsychological tests (Finlayson et al., 1977). Groups of subjects were equated for level of education (+/- 1 year) and IQ (+/- 10 points). In order to estimate IQ, subjects were given the PPVT-R during the screening phase of the study. The PPVT-R is designed to assess one-word receptive vocabulary and is highly correlated with the Wechsler Adult

Intelligence Scale (WAIS) Full Scale IQ score. The test requires the subject to choose one of four items displayed on a card as depicting the word spoken by the examiner. After five training items, 175 items of increasing difficulty can be given, but usually only 35 to 45 items need to be administered if a suitable beginning point is chosen. A basal point is established when the subject provides six consecutive correct responses. Subjects are given credit for all items below the basal point. Testing is discontinued when six out of eight consecutive items are failed (Dunn & Dunn, 1981). Two alternate forms (L and M) are available. Form L was used in the present study.

The score on the PPVT is determined by counting the number of items passed, including the items prior to the basal point. The manual allows translation of these scores into "age equivalents" (previously called "mental age"), standard score equivalents (previously called "IQ"), stanines, and percentiles (Dunn & Dunn, 1981; Spreen & Strauss, 1991). PPVT standard scores were compared with WAIS standard scores. The correlations with the WAIS Verbal Scale ranged from .21 to .91, with a median of .71; with WAIS Full Scale scores, correlations ranged from .17 to .92, with a median of .72. The median correlation with WAIS Performance Scale scores was .65 (Dunn & Dunn, 1981).

The PPVT has been standardized on a sample of people considered representative of the United States population ranging in age from 2.5 to 40 years. Split-half reliability has been reported as ranging from .61 to .88 in

children and adolescents, and as .82 for Form L in adults (Dunn, L.M. & Dunn, L.M., 1981).

Dependent Variables

Selective Reminding Test (SRT)

The SRT (Buschke, 1973) was used to assess new learning and memory. The SRT has been used extensively to investigate verbal free recall in patients with memory disorder, and has proven useful in elucidating the memory deficit associated with alcoholic Korsakoff's syndrome (Buschke & Fuld, 1974), dementia of the Alzheimer type, and traumatic brain injury (Hannay & Levin, 1985; Levin et al., 1982; Levin & Grossman, 1976; Paniak, Silver, Finlayson, & Tuff, 1989). Head injury adversely affects the ability to place newly learned material into long-term memory (Shapiro & Sacchetti, 1993), and the ability to retrieve material from memory once stored (Gronwall & Wrightson, 1981). PCS is associated with exacerbated new learning and memory difficulties that result from MHI (Bohnen et al., 1992; Gronwall & Wrightson, 1974). Additionally, sleep deprivation impedes new learning and retrieval ability (Bonnet, 1994; Elkin & Murray, 1974; Gieseking et al., 1957). Thus, the compound effects of head injury, PCS, and sleep deprivation were expected to maximally adversely affect performance on the SRT.

The SRT involves reading the subject a list of words and then having the subject recall as many list words as possible, in any order. For each subsequent learning trial, the examiner selectively presents only those items that were not recalled on the immediately preceding trial (Buschke, 1973;

Buschke & Fuld, 1974), though subjects are expected to attempt to recall all items. The test consists of 12 unrelated words. Lists of this length are referred to as "super span" lists, as they exceed the seven (plus or minus two) items that subjects can typically hold in short-term memory. Learning the complete list requires repeated presentations; thus words are presented over 12 selective reminding trials, or until the subject is able to recall the entire list on three consecutive trials.

Buschke pointed out that the SRT primarily differs from other recall procedures by selectively presenting only those items which were not recalled during the immediately preceding trial. By measuring recall of items which are not presented on a given trial, the procedure distinguishes between retrieval from long-term storage and short-term recall. The short-term memory component of the SRT normally diminishes across trials as the subject requires less reminding and retrieves more information from long-term storage (Hannay & Levin, 1985).

The SRT has been modified for various patient groups by varying the length of the word list (e.g., 6 to 20 words), and adding a multiple-choice recognition trial (list word, homonym, synonym, unrelated ditsractor), and a 30-minute delayed-recall trial following the standard test (Levin et al., 1982). Hannay and Levin (1985) found that of the four SRT forms available, Form 1 is significantly more difficult than Forms 2, 3, and 4, which did not differ from each other. Further, performance on the first administration of the SRT was found to be poorer than on subsequent administrations. Performance on the second,

third, and fourth administrations was similar. Thus, researchers are encouraged to obtain baseline scores to insure a stable level of pre-treatment performance. Test-retest reliability for the measure has ranged from .48 to .65 (Spreen & Strauss, 1991).

The SRT instructions read as follows: "This test is to see how quickly you can learn a list of words. I am going to read you a list of words. I want you to listen carefully because, when I stop, I want you to tell me as many of the words as you can recall. The words do not have to be in any particular order. When you have given me all the words that you can recall, I will tell you the words that you didn't give me from the list; then I want you to give me the entire list all over again. We do this twelve times, and each time I want you to try to give me all twelve words" .

In the present study, the list of words was read at a rate of one word per two seconds. The words were always presented in the same order, beginning with the top of the list and working to the bottom. Upon completion of the first trial, subsequent list presentations omitted the words that were recalled correctly on the preceding trial. When a subject was able to correctly recall all 12 words on three consecutive trials, the test was discontinued, but scored as if all trials (maximum of 12 trials) had been given with 100 percent recall following the three consecutive repetitions. If the subject recalled a word not on the list, the subject was informed, and the extra word(s) noted. The total number of words on the list was not disclosed. For the delayed recall trial, presented after a 30-minute delay, the subject was asked to recall as many

words as possible, in any order, from the most recently presented list. The multiple-choice recognition trial was given even if the subject correctly recalled the entire list on the delayed recall trial. The SRT requires approximately 45 minutes to administer: 10 minutes to administer the learning trials, a 30-minute delay, and approximately 5 minutes to administer the delayed recall and recognition trials (Spreen & Strauss, 1991).

Scoring of the SRT is designed to determine the number of words a subject is capable of integrating into long-term storage (LTS). When a word is recalled on two consecutive trials, it is assumed to have entered LTS on the first of these trials. This inference is derived from the observation that the subject recalled a word which had not been presented by the examiner on that trial (i.e., no reminder was given on trial *n* for a word correctly recalled on trial *n* -1). Once a word has entered LTS, it is considered to be in permanent storage. Consequently, the word is scored as LTS on all following trials irrespective of the subject's subsequent recall. When a subject begins to recall a word in LTS consistently on all subsequent trials, it is also scored as consistent long-term retrieval (CLTR) (Hannay & Levin, 1985).

In the present study, the SRT was administered three times. The first administration constituted a learning trial. The following two administrations represented pre- and post-sleep deprivation trials, respectively. Forms 2, 3, and 4 of the standard 12-word version of the SRT were used in the present study. The 12-word SRT is the version most often used with adults in clinical and research settings. Further, alternate forms and normative data are

available for this version (Spreen & Strauss, 1991). The 12-trial immediate recall aspect of the SRT were followed by a delayed (30-minutes) recall and a delayed recognition trial. Five SRT scores were compared between groups: Immediate Recall Score, defined as the total number of words recalled across immediate recall trials; LTS Score, defined as total number of words integrated into LTS across immediate recall trials; CLTR score, defined as the total number of words recalled consistently on all subsequent trials following; Delayed Recall Score; and Delayed Recognition Score. Head-injured and uninjured control subjects were expected to perform at a near equal level prior to sleep deprivation. While overall performance of both groups was expected to degrade following sleep deprivation, head-injured subjects were expected to perform significantly worse than uninjured controls on all aspects of the SRT.

Paced Auditory Serial Addition Test (PASAT)

The Paced Auditory Serial Addition Test (PASAT) was used to assess rate of information processing and sustained attention. MHI adversely affects information processing ability in that it decreases processing speed, thereby limiting the amount of material that can be processed swiftly and efficiently (Gronwall & Wrightson, 1981). PCS is associated with exacerbated information processing deficits following MHI (Gronwall & Wrightson, 1974). Additionally, sleep deprivation results in impaired information processing (Bonnet, 1994; Broadbent, 1971). Thus, the compound effects of head injury, PCS, and sleep deprivation were expected to maximally adversely affect performance on the PASAT.

The PASAT was devised by Gronwall and colleagues (Gronwall, 1977; Gronwall & Wrightson, 1974) to provide an estimate of the subject's rate of information processing and the amount of information that can be handled at one time. The PASAT has been shown to be sensitive to the effects of MHI (Gronwall & Sampson, 1974; Gronwall & Wrightson, 1975), to relate to the patient's experience of symptoms, and to indicate readiness to return to work (Gronwall, 1977). Although it is a better predictor of subsequent memory difficulties than post-traumatic amnesia (Gronwall, 1981), the PASAT is not primarily a memory task (Gronwall & Wrightson, 1981). Further, Gronwall claims that although it is a cognitive task, there is only a small correlation with arithmetic ability (.28) and general intelligence (.28) (Spreen & Strauss, 1991).

During the administration of the PASAT, a pre-recorded tape delivered a random series of 60 numbers from 1 though 9. The subject was instructed to add pairs of numbers such that each number is added to the one immediately preceding it: The second number is added to the first, the third to the second, the fourth to the third, and so on. For example, if given the numbers "1, 9, " the answer is "10"; if the next number is "4", this is added to the previous number "9" to give the answer "13"; and so on. If a subject was unable to understand the task after listening to the recorded instructions, additional instructions were read to him or her. If a subject was still unable to understand the task after receiving recorded and oral instructions, a written example was provided. A practice trial was given, followed by the presentation of the first PASAT trial. After the first trial, subjects were told that rate of presentation would increase

on subsequent trials. Instructions were not repeated unless the subject demonstrated on the paced practice trial that he or she had forgotten what to do (Spreen & Strauss, 1991). If the subject lost his or her place during a trial, he or she was told to continue the task by adding the next two consecutive numbers and proceeding onward.

The same 60 numbers were presented in four different trials, each differing in the rate of digit presentation (one digit every 2.4, 2.0, 1.6, and 1.2 seconds). The PASAT thus increases processing demands by increasing the speed of stimulus input. The duration of each spoken digit is about 0.4 second (Spreen & Strauss, 1991). The subject was required to comprehend the auditory input, add two numbers together, respond verbally, inhibit encoding of one's own response while attending to the next stimulus in a series, and perform at an externally determined pace (Spreen & Strauss, 1991). Information processing ability was inadequate if the number of items demanding simultaneous attention was too great or if the rate of processing was too slow (Gronwall & Wrightson, 1974).

When scoring the PASAT, the number of correct and incorrect responses per trial (i.e., at each of the four pacing rates) were recorded. To be correct, a response must be made before presentation of the next stimulus. The maximum score per trial is 60. The cutoff point for impairment on the PASAT is one standard deviation below the mean of control subjects (Gronwall, 1977).

As with the SRT, significant practice effects have been noted with the use of the PASAT (Gronwall, 1977). Normal subjects who are given the PASAT on two occasions, spaced one week apart, perform about 6 points higher on the second administration (Stuss et al., 1987). After the second presentation, practice effects tend to be minimal (Gronwall, 1977). The PASAT's split-half reliability is .96, implying high internal consistency (Spreen & Strauss, 1991).

In the present study, the PASAT was administered three times. The first administration constituted a learning trial. The following two administrations represented pre- and post-sleep deprivation trials, respectively. The PASAT total score (eg.g, the overall number of correct responses across four trials) was examined between groups. Based on previous research (Betz et al., unpublished manuscript), the groups were expected to perform at nearly the same level at baseline. While overall performance of both groups was expected to degrade following sleep deprivation, head-injured subjects were expected to perform significantly worse than uninjured controls on all aspects of the PASAT.

Laboratory Procedures

Subjects were instructed to obtain a minimum of six and a maximum of eight hours of sleep the night before the experiment. Further, they were instructed to abstain from drinking alcoholic or caffeinated beverages 24 hours prior participating in the study. Subjects reported to the neuropsychology laboratory at Louisiana State University at 08:00 hours on the morning of the

experiment (Day 1). Upon arrival at the laboratory, the PASAT and the SRT were administered as a learning trial. Presentation of the SRT and the PASAT was counterbalanced within subjects to control for order effects. All subjects were required to remain in the laboratory under the supervision of the experimenter and/or a research assistant. They were not allowed to nap or to consume caffeine. However, they were permitted to read, watch television, etc. until the next test period.

Baseline data was collected at 20:00 hours on Day 1, at which point the PASAT and SRT were readministered. Alternate forms of the SRT were used to prevent memorization of stimuli which could result in artificially inflated scores. The order of presentation of SRT forms was randomized. Once baseline data had been collected, non-sleep deprived subjects were released and told to return to the neuropsychology laboratory at 08:00 hours the following morning (Day 2). They were instructed to again obtain a minimum of six and a maximum of eight hours of sleep and abstain from drinking alcoholic or caffeinated beverages. Sleep deprived subjects remained supervised in the laboratory and were required to remain awake until 22:00 hours the following evening.

Non-sleep deprived and sleep deprived subjects were retested at 20:00 hours on Day 2. Post-sleep deprivation data was collected exactly 24 hours after baseline data (20:00 hours on Day 2) so as to control for potentially confounding effects of circadian rhythmicity (Babkoff, Temir, & Mikulincer, 1991). Thus, following 36 hours of sleep deprivation, all subjects completed

the Driver Risk Index (DRI) and the Driver Performance Test (DPT). Prior research has shown that the effects of sleep deprivation on cognitive performance are best detected after 30 to 50 minutes of testing (Donnell, 1969). Thus, the DRI and DPT were administered as "filler tasks" to prolong the time of cognitive engagement. The DRI and DPT were administered to all subjects simultaneously. Both measures required the subject to watch a video tape which depicts various traffic scenes recorded from a camera stationed on the dashboard of a "driver car". The DPT requires subjects to observe 50 traffic scenes. Following each scene, the subject must decide which of four options was the most important aspect of the traffic scene to attend to, as far as driver awareness and safety are concerned. The DRI requires subjects to observe 40 different traffic scenes. During each scene the driver makes a comment relevant to the scene such as, "I have sufficient time and space to pass." The subject is required to agree or disagree with the driver's comment by circling "agree" or "disagree" his of her answer sheet. Administration of the DRI and DPT required 50 minutes, after which the PASAT and the SRT were re-administered (20:00 hours). When testing was completed, subjects were debriefed and released (approximately 22:00 hours, Day 2). Sleep deprived subjects were not allowed to drive themselves home upon completion of the study. Thus, sleep deprived subjects were asked to arrange for transportation when recruited to participate in the study. If a subject was unable to arrange for transportation, the experimenter arranged for the subject to be driven home. A schedule of the experimental protocol is provided in Table 1.

Table 1. Experimental Protocol

Day 1 (Saturday)	
08:00 - 09:00	Subjects Arrive: schedule is discussed.
09:00 - 10:00	PASAT 1 and SRT 1 administered
10:00 - 12:30	Free Time: Subjects are allowed to read, watch movies, work on coursework etc.
12:30 - 13:30	Lunch Provided.
13:30 - 17:30	Free Time
17:30 - 18:30	Dinner Provided
18:30 - 20:00	Free Time
20:00 - 22:00	PASAT 2 and SRT 2 administered
22:00	Non-sleep deprived subjects released for the evening.
22:00 - 00:00	Free Time
Day 2 (Sunday)	
00:00 - 07:00	Free Time: subjects not allowed to sleep
07:00 - 08:00	Breakfast Provided/Non-sleep deprived subjects return
08:00 - 12:00	Free Time
12:00 - 13:00	Lunch Provided
13:00 - 17:30	Free Time
17:30 - 18:30	Dinner Provided
19:00 - 19:50	DRI and DPT presented
20:00 - 22:00	PASAT 3 and SRT 3 administered
22:00	Subjects are debriefed and released

EXPERIMENTAL HYPOTHESES

The following hypotheses were proposed:

1. The performance of non-sleep deprived head-injured and uninjured control subjects was not expected to differ on any aspect of the SRT or the PASAT across administrations.
2. Prior to sleep deprivation, the performance of head-injured and uninjured control subjects was not expected to differ on any aspect of the SRT or the PASAT.
3. Following sleep deprivation, performance of both head-injured and uninjured control subjects was expected to decline on all aspects of the SRT and the PASAT.
4. (a) A greater difference between pre- and post-sleep deprivation performance was expected to occur among head-injured subjects on the following aspects of the SRT: (a) number of words recalled across immediate recall trials; (b) number of words integrated into LTS; (c) number of words consistently recalled (CLTR); (d) number of words recalled after a 30-minute delay; and (d) number of words recognized after a 30-minute delay.

4. (b) A greater difference between pre- and post-sleep deprivation performance was expected to occur among head-injured subjects on the PASAT in that MHI subjects were expected to obtain a lower overall number of correct responses.

RESULTS

Between groups univariate analyses of variance (ANOVAs) were performed on subject demographic data to insure group comparability. Demographic variables subjected to an ANOVA included: age, years of college, PCSC total score, PPVT-R score, BDI score, STAI-state score, and STAI-trait score. For head injured subjects, the number of head injuries, and month since last injury were also subjected to ANOVAS. Sample characteristics are presented in Table 2. Group means were not found to differ significantly on any of the aforementioned subject variables: Group means and standard deviations are provided in Table 3. Results of univariate F-tests for subject variables are presented in Table 4.

A total of six dependent variables were extracted from the dependent measures, five variables from the SRT and one from the PASAT. Dependent variables were defined in the following manner: (1) Total Recall was defined as the total number of words recalled across SRT immediate recall trials; (2) Long-Term Storage (LTS) was defined as the total number of SRT words integrated into LTS across immediate recall trials; a word must be freely recalled on two consecutive trials to be included in the LTS score; (3) Consistent Total Long-Term Recall (CLTR) was defined as the total number of SRT words consistently recalled across trials; a word is said to enter CLTR on the first of uninterrupted successful recall trials; (4) Delayed Recall was defined as the total number of SRT words recalled after a 30 minute delay; (5) Delayed

Table 2: Sample Characteristics (n = 60): Means and Standard Deviations

Measure	HI/SD	HI/NSD	NHI/SD	NHI/NSD
Age	20.40 (2.35)	21.13 (4.68)	22.00 (5.69)	22.60 (6.50)
Years College	2.40 (.91)	2.20 (1.26)	2.66 (1.05)	3.07 (1.03)
# of Injuries	2.13 (1.24)	1.80 (.94)		
Months Post-Injury	55.53 (50.39)	49.80 (50.41)		
PCSC 2 months	66.33 (10.76)	70.00 (13.19)	62.53 (7.57)	67.27 (9.72)
BDI	5.66 (5.62)	6.00 (3.55)	5.60 (5.28)	6.13 (3.46)
STAI - state	49.80 (10.82)	47.60 (8.78)	46.26 (8.29)	50.73 (7.88)
STAI - trait	50.00 (9.87)	50.87 (7.41)	51.86 (9.17)	56.07 (7.74)
PPVT - R	106.47 (11.13)	107.27 (10.89)	107.20 (14.25)	109.73 (13.64)

- HI/SD = Head injured/Sleep Deprived
- HI/NSD = Head Injured/Non-Sleep Deprived
- NHI/SD = Non-Head Injured/Sleep Deprived
- NHI/NSD = Non-Head Injured/Non-Sleep Deprived

Table 3. Univariate F-tests conducted on subject variables with (3,56) degrees of freedom.

Variable	Univariate F	p Value
Age	.59	.62
Years of College	1.83	.1513
PCSC 2 Months	.94	.43
BDI	.05	.99
STAI-state	.77	.52
STAI-trait	1.46	.23
PPVT-R	.19	.90
Number of Head Injuries		
Months Since Injury	.63	.80

Recognition was defined as the total number of SRT words recognized after a 30 minute delay; (6) the PASAT Total Score was defined as the total number of correct PASAT responses recorded across four pacing speeds. Independent variables consisted of the following subject variables: Head injury status (injured or uninjured) and sleep deprivation status (sleep deprived or non-sleep deprived).

A multivariate analysis of variance (MANOVA) was employed to determine if performance on the SRT differed between groups at baseline (pre-deprivation trial). Thus, a 2 (head injury status) x 2 (sleep status) MANOVA was performed on the five baseline SRT scores. Dependent variables were entered into the model simultaneously. With the use of Wilk's Criterion, the groups (head injured/sleep deprived, head injured/non-sleep deprived, uninjured/sleep deprived, and uninjured/non-sleep deprived) were not found to differ significantly on the combined dependent variables [F = .268 (5, 52), p < .928). No significant main effects were found for head injury or sleep deprivation. Further, no significant interactions were noted between head injury and sleep deprivation.

A between groups ANOVA performed on baseline PASAT total score data was not significant [F = .846 (2, 59), p < .435). No main effects were noted for head injury or sleep deprivation. Further, no significant interactions were noted between head injury and sleep deprivation. Means and standard deviations for the five baseline SRT variables and the baseline PASAT total score are presented Table 4.

Table 4. Group means and standard deviations for baseline SRT and PASAT dependent variables (* = $p < .05$).

Variable	HI/SD	HI/NSD	NHI/SD	NHI/NSD
*SRT Delayed Recall	8.80 (2.80)	10.87 (1.41)	8.53 (3.50)	9.53 (1.88)
SRT Delayed Recognition	11.53 (.64)	11.57 (.82)	11.53 (1.06)	11.60 (.83)
SRT Long Tern Storage	106.87 (23.14)	114.53 (19.62)	103.33 (17.34)	109.67 (16.01)
SRT Consistent Long-Term Recall	78.87 (39.85)	87.93 (37.77)	67.40 (30.01)	75.80 (31.12)
SRT Total Recall	113.40 (16.91)	118.80 (16.08)	109.20 (12.87)	111.93 (12.68)
PASAT Total Score	156.93 (45.23)	153.40 (39.59)	166.87 (37.05)	170.93 (41.30)

- HI/SD = Head Injured/Sleep Deprived
- HI/NSD = Head Injured/Non-Sleep Deprived
- NHI/SD = Non-Head Injured/Non-Sleep Deprived
- NHI/NSD = Non-Head Injured/Non-Sleep Deprived

A repeated measures MANOVA was performed on the five SRT dependent variables to assess for the effects over time. Thus, scores on the five SRT variables were compared across the three sampling times (i.e., learning trial, pre-sleep deprivation trial, post-sleep deprivation trial). The difference between Trials 2 (pre-deprivation) and 3 (post-deprivation) was of greatest interest. The SRT variables were entered into the model simultaneously. With the use of the Wilk's criterion, a significant effect was found for time [F (10, 216) = 2.46, $p < .008$], but no significant effect was found for head injury or sleep deprivation. No interactions were found among head injury, sleep deprivation, and time. Univariate analyses showed a significant effect across groups for time on the SRT Delayed Recognition measure [F (2, 112) = 6.38, $p < .002$], and the SRT Delayed Recall measure [F (2, 112) = 6.79, $p < .002$]. The results of univariate ANOVAs are provided in Table 5. As illustrated in Figure 1, performance on the Delayed Recognition aspect of the SRT progressively declined from the Learning Trial (Trial 1) to the post-sleep deprivation trial (Trial 3) across groups. Figure 2 illustrates that performance on the Delayed Recall portion of the SRT also declined across groups from Trial 1 to Trial 3. Post-hoc analyses were conducted using the Tukey's Honestly Significant Difference (HSD) test. SRT Delayed Recognition and Delayed Recall scores for all groups had to be combined for group means to be compared across time (Trials 1, 2, and 3). The Tukey's HSD test confirmed the difference in SRT Delayed Recognition combined means: The combined

means were found to differ significantly between Trial 1 and Trial 2, and Trial 1 and Trial 3 [F = 5.49, (2, 177), p < .0049], but not between Trial 2 and Trial 3. Results of the Tukey's HSD test found that SRT Delayed Recall combined means did not differ significantly across trials. The Tukey's HSD test was performed again on the SRT Delayed Recognition and Delayed Recall data without collapsing across group means, i.e., Delayed Recall and Delayed Recognition data were examined separately for each group across the three trials. No significant differences were found between trials for either Delayed Recognition or Delayed Recall data.

A repeated measures ANOVA was also performed on PASAT total score data. A main effect was found for time [F (2,112) = 50.17, p < .0001). Post-hoc analyses conducted using Tukey's HSD test confirmed the difference between Trial 1 and Trial 2, and Trial 1 and Trial 3 of the PASAT Total Score data, when data was collapsed across groups [F = 10.17 (2, 177), p < .0001]. No significant difference was found between Trial 2 and Trial 3. The Tukey's HSD test was performed again without collapsing across group means. Thus, PASAT Total Score data were examined separately for each group across three trials. This manipulation resulted in a significant difference found between the Learning Trial (Trial 1) and the post-sleep deprivation trial (Trial 3) [F (2, 42) = 3.74, p < .03], but for the uninjured/non-sleep deprived group, only.

A between groups multivariate analysis of covariance (MANCOVA) was used to assess the difference between groups in pre- and post-sleep deprivation performance change. Thus, a 2 (head injury) by 2 (sleep

deprivation status) MANCOVA was performed analyzing the five SRT post-sleep deprivation (Trial 3) scores, with pre-sleep deprivation (Trial 2) scores representing the covariates. With the use of the Wilk's criterion, groups were not found to differ significantly on the combined dependent variables. No main effects were found for head injury or sleep deprivation. Further, no interactions were found between head injury and sleep deprivation. A between groups analysis of covariance (ANCOVA) was performed on post-sleep deprivation PASAT Total Score data using the pre-sleep deprivation total score as the covariate. Post-sleep deprivation performance was not found to differ significantly between groups. No main effects were noted for head injury or sleep deprivation. Further, no interactions were found between head injury and sleep deprivation.

Table 5. Univariate F-tests following repeated measures MANOVA and repeated measures ANOVA with (2,112) degrees of freedom.

Variable	F value	Significance of F
PASAT Total Score	52.76	.0001
SRT Delayed Recognition	6.70	.002
SRT Delayed Recognition	6.38	.002
SRT Total Recall	2.16	.12
SRT Long Term Storage	1.06	.35
SRT Consistent Long-Term Recall	.98	.38

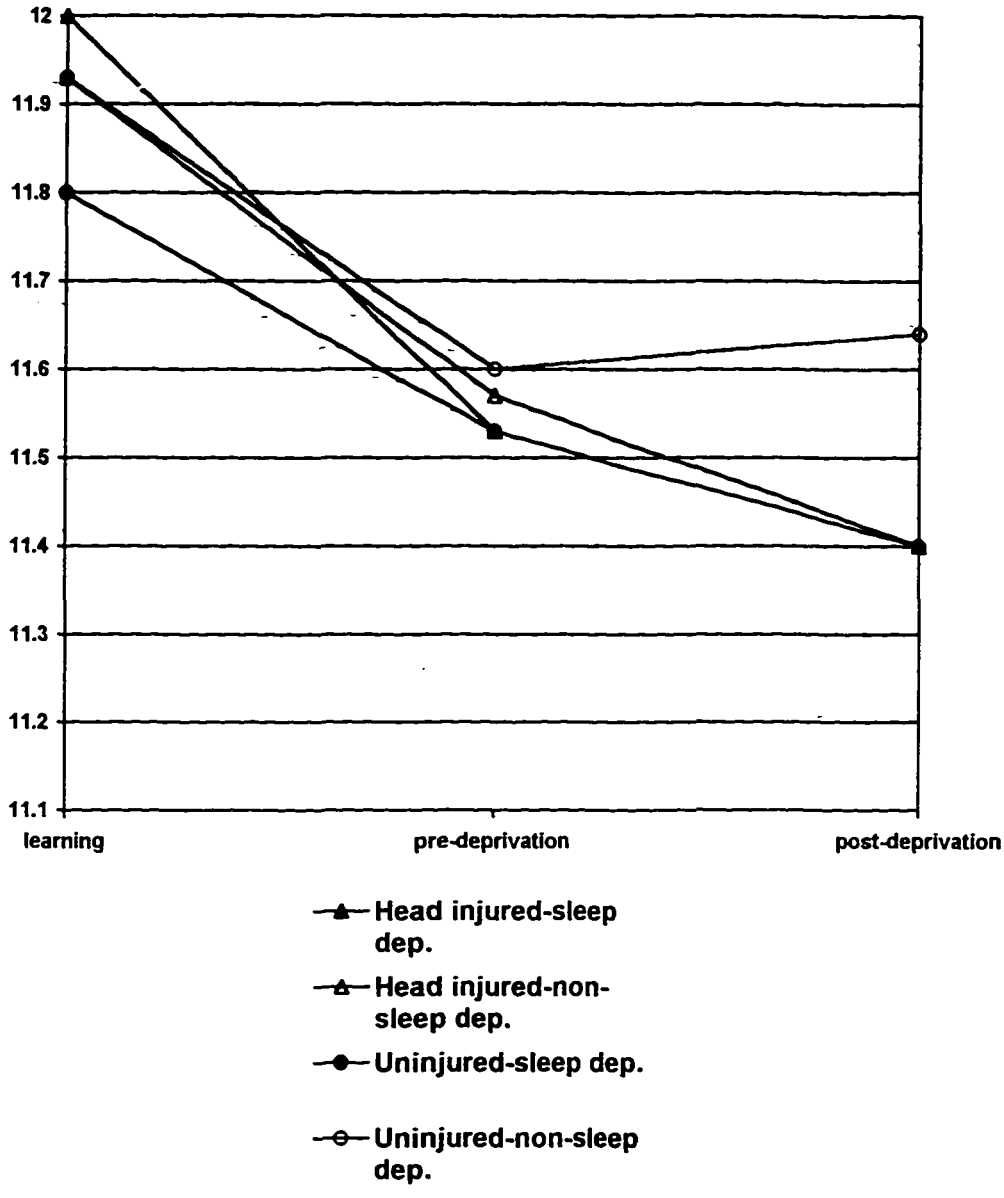

Figure 1: The number of words recognized after a 30-minute delay across three presentations of the SRT.

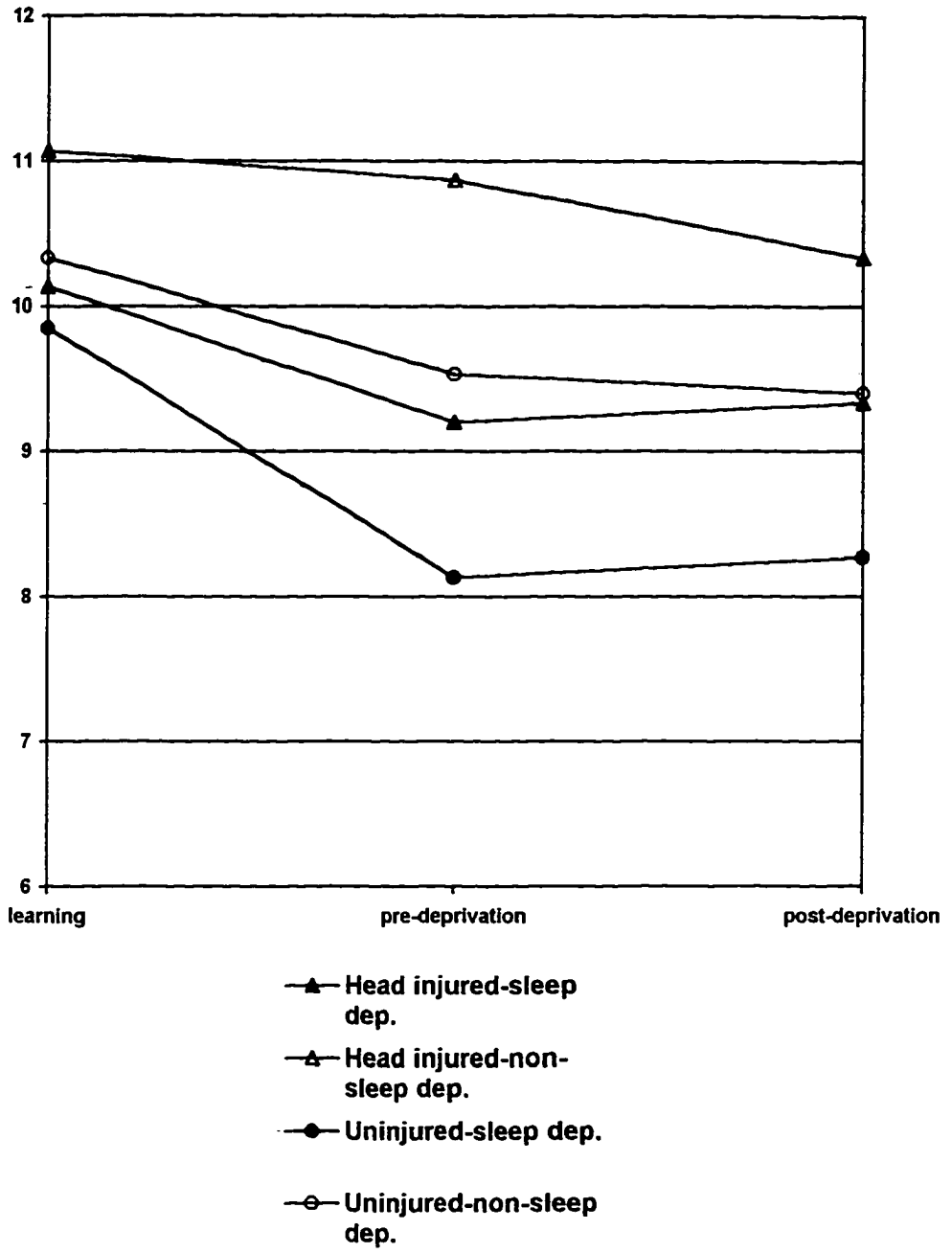

Figure 2. The number of words recalled after a 30-minute delay across three SRT presentations.

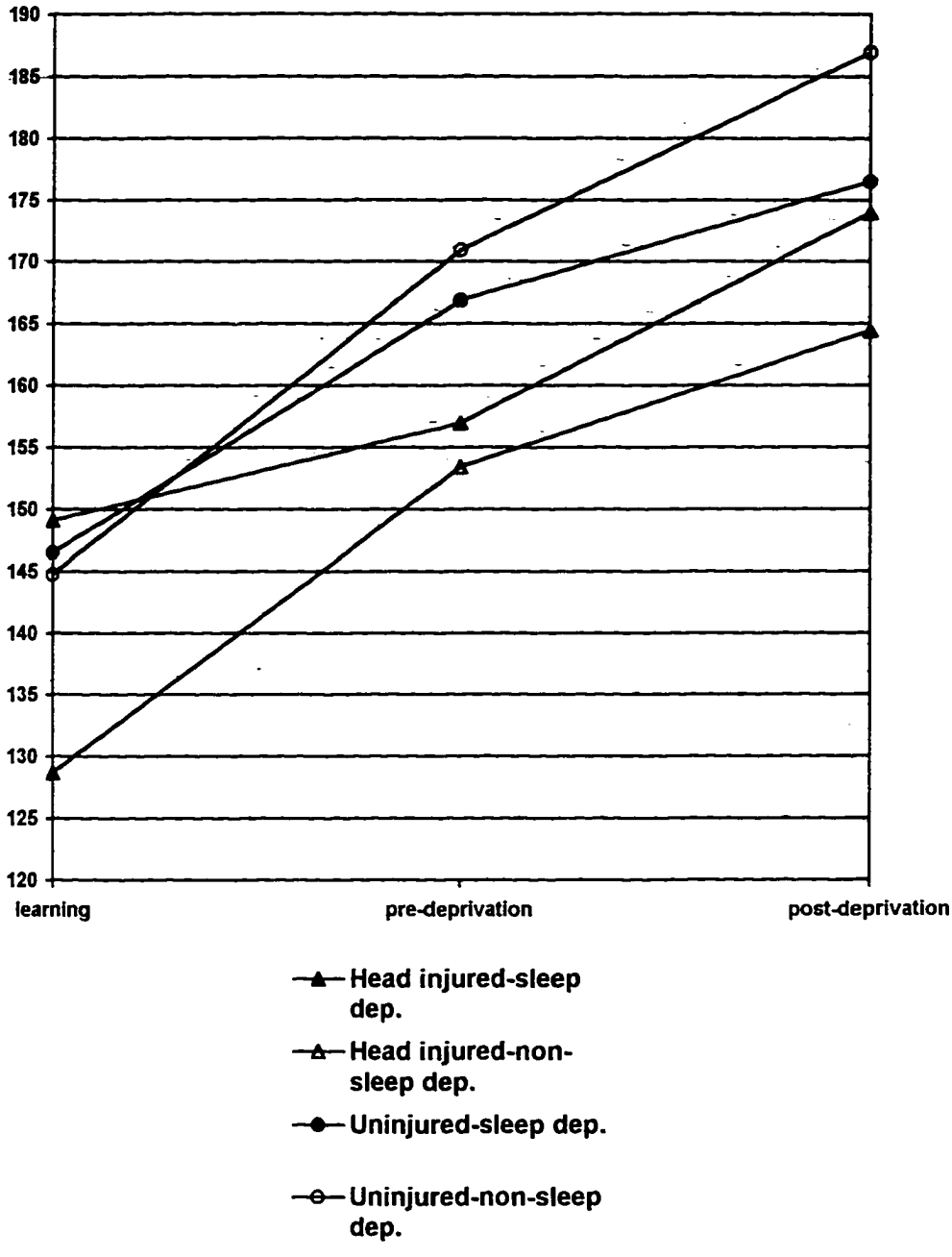

Figure 3. Total number of correct responses across three PASAT presentations.

DISCUSSION

Results of the current investigation revealed no significant differences between groups (head injured/sleep deprived, head injured/non-sleep deprived, uninjured/sleep deprived, and uninjured/non-sleep deprived) for the PASAT Total Score variable after the learning trial (Trial 1) and just before sleep deprivation (Trial 2). Similarly, no group differences were found on the five SRT variables at Trial 2. Thus, one learning trial was sufficient to familiarize subjects with the PASAT and SRT and produce comparable baseline levels of performance.

A significant effect for time was found for the SRT Delayed Recognition data, with level of performance declining across groups between Trial 1 and Trial 2, and Trial 1 and Trial 3. A significant effect for time was found for the PASAT Total Score variable, with performance improving across groups from Trial 1 to Trial 2, and from Trial 2 to Trial 3. However, no significant differences were found between groups for the five SRT variables and the PASAT Total Score following 36 hours of sleep deprivation or following 36 hours of normal activity and sleep. Lack of group differences following sleep deprivation was unexpected, as it was hypothesized that sleep-deprived subjects would perform worse than non-sleep deprived controls post-sleep deprivation. It was further hypothesized that head injured subjects would perform worse than uninjured subjects following sleep deprivation, as the effects of head injury and sleep deprivation were expected to be additive. The

following sections will offer some plausible reasons that the experimental hypotheses were not supported by the obtained data.

Previous research has documented deficits in information processing (Boll, 1993; Gentilini et al., 1985, Gronwall & Wrightson, 1981; Gronwall & Wrightson, 1974; Stuss et al., 1985), and new learning and memory (Gronwall & Wrightson, 1981; Levin et al., 1988; Shapiro & Sacchetti, 1993) following MHI. Injury-related cognitive deficits have been noted to remit between one month (Gronwall & Wrightson, 1974; Hugenholtz et al., 1988) and 6 months (Dikman et al., 1983; Jennett et al., 1981; Levin et al., 1987) following MHI. Once recovery is complete, the cognitive functioning of individuals who have sustained a MHI is typically comparable to uninjured controls. However, research has shown that cognitive deficits emerge among mildly head injured individuals under conditions of hypoxic stress (Ewing et al., 1981). The hypothesis of the present study was that lack of sleep would potentiate subtle injury-related cognitive deficits thought to exist among head-injured subjects. It is possible, however, that cognitive deficits were not elicited post-sleep deprivation among MHI subjects in the present study due to the minor nature of the injury sustained by a majority of subjects in the sample tested.

Previously cited studies on the cognitive effects of MHI investigated a subject sample that, although classified as mildly injured, still may have been more severely injured than subjects in the current study. Research conducted on victims of MHI typically has investigated cohorts in which the majority of subjects reported experiencing a brief loss of consciousness, as opposed to

merely being stunned or dazed following injury (Arcacia & Gualtieri, 1993; Bohnen et al., 1992; Clooney-Hogansen et al., 1984; Dikmen et al., 1987). Further, most studies of MHI examined subjects who sought medical treatment after their injury and were followed as outpatients in neurology clinics (Arcacia & Gualtieri, 1993; Mittenberg et al., 1992; Clooney-Hogansen et al., 1984), or who were hospitalized for a period of not longer than three days (Bohnen et al., 1992; Dikmen et al., 1987; Ewing et al., 1981; Gronwall & Wrightson, 1974; Leininger et al., 1989; Levin et al., 1987; Shapiro & Sacchetti, 1993). Though head injury in the aforementioned studies was classified as mild and uncomplicated (e.g., no radiological abnormalities, skull fracture, or hematoma), subjects experienced loss of consciousness, and were hospitalized and/or followed as outpatients, which implies that their injuries fell at the more severe end of the MHI continuum.

In the present study, however, a majority of subjects who had sustained a MHI did not experience a loss of consciousness (87%), and did not seek medical treatment (72%) either immediately following the injury or at some later point. Given that subjects were not rendered unconscious, they experienced what is classified as mild concussion. The effects of mild concussion are reported to be less severe than classical cerebral concussion, which involves a period of unconsciousness (Genarelli, 1993; Williams et al., 1990). The statement is based on the premise that a blow to the head capable of rendering one unconscious is of greater velocity than an insult that results in

the victim being dazed. As velocity increases, so does of the extent of diffuse brain injury (Genarelli, 1993; Genareli et al., 1982).

Finally, studies that have reported the means by which MHI occurred listed motor vehicle accidents (MVA) as the primary cause of injury (Bohnen et al., 1992; Leininger et al., 1989; Rimel et al., 1982; Stuss et al., 1985). The acceleration/deceleration component inherent in MVAs results in diffuse brain injury, the severity of which is directly proportional to the velocity at which the head moves from rest or to rest during the injury sequence (Genarelli et al., 1985; Genarelli et al., 1982). It is likely that one who sustained an injury in a MVA was exposed to greater acceleration/deceleration forces than one who was injured, for example, during a fall or while participating in a sporting event, and thus incurred more extensive diffuse brain injury.

In the present study, a majority of subjects sustained sporting injuries (57%), which were followed in frequency by falls (21%), MVAs (20%), and assaults (2%). While acceleration/decereration forces are a factor in sporting injuries and falls, it is likely that the velocity with which one is hit by another player or with which one hits the ground is significantly less than that experienced in a MVA. Given that 78 percent of subjects in the present study were injured during a sporting event or a fall, it is thus likely that their injuries were less severe than injuries sustained during a MVA.

Two additional factors may help to explain why the injuries of subjects in the current study may be less severe those generally sustained by subjects most often included in investigations of MHI. First, subjects were relatively

young when injured (mean age of injury was 16 years old). Previous research has shown that prognosis following MHI varies with age, with younger persons recovering more quickly and completely than older persons (Barth et al., 1983; Rutherford et al., 1979; Wrightson & Gronwall, 1981). Second, as noted in Gouvier et al. (1992), the use of mildly head injured college students as subjects may reduce the likelihood that cognitive deficits will be noted, as it is most likely that injured subjects who have been able to remain enrolled in classes at a university have substantially recovered from their injuries.

Endorsement of PCS symptoms appears to covary with neuro-psychological impairment, i.e., MHI patients who present with such symptoms have been found to have greater deficits in information processing and new learning (Leininger et al., 1990; Gronwall & Wrightson, 1974). In the present study, preliminary mass screening revealed that head injured and uninjured subjects from the available undergraduate population endorsed symptoms of PCS at a level considerably lower than subjects in the Gouvier et al. (1992) study, in which the PCSC was administered to college undergraduates. Thus, the PCSC Total Score cutoff had to be defined by a median split rather than the empirically determined cutoff scores originally proposed. Subjects who scored above the 50^{th} percentile (PCSC Total Score of $\geq$ 57 for MHI subjects with PCS, and $\geq$ 56 for uninjured subjects with PCS) were included in the study. PCSC Total Score means obtained for PCS subjects in the present study (head injured group mean = 68.16; uninjured group mean = 64.90) were consistent with those obtained by Gouvier et a. (1992) for subjects without PCS (head

injured group mean = 71.35; uninjured group mean = 64.85). Further, the PCSC group means for injured and uninjured subjects in the present study did not exceed the Gouvier et al. (1992) group means of subjects without PCS by one and one-half standard deviations, as originally proposed. Finally, as might be expected, the MHI and uninjured PCS groups did not differ significantly on PCSC Total Score.

Several factors may have resulted in the lack of deficits found among PCS-reporting subjects in the present study. Although subjects endorsed symptoms of PCS at a level comparable to that of previously assessed head injured subjects in one study (Gouvier et al., 1992), the level of endorsement in the present study was not in the clinically significant range. While subjects in the current study endorsed a level of PCS that was significantly greater than 50 percent of the population screened, it is likely that symptoms were not of great enough frequency, intensity, or duration to warrant the classification of PCS. It is thus doubtful that the PCS variable could have contributed anything to cause performance decrements in these subjects or differences in performance among the groups.

A significant relation has been found between the report of PCS symptoms immediately post-injury and complaint of neuropsychological deficits (e.g., difficulty concentrating, memory problems, etc.) several months later (Leininger et al., 1990). If one does not experience PCS symptoms within one month of MHI, the chances that complaints associated with PCS will ever be voiced decrease significantly (Binder, 1986; Levin, 1989). Although unknown,

it is unlikely subjects in the current study were symptomatic post-injury because so few of them required follow-up care (hospitalization or outpatient care) after their accidents.

Research conducted by Gouvier et al. (1992) found that endorsement of above average stress levels covaried with an increase in reported symptoms on the PCSC. Thus, it is possible that subjects in the current study were endorsing symptoms caused by current life stressors (e.g., college, family, work, etc.) and may not truly have been experiencing PCS. If this was the case, the same relation between PCS and performance on neuropsychological measures cannot be expected.

Sleep deprivation has been found to adversely affect information processing and memory (Bonnet, 1994; Geisking et al., 1957; Lingenfelser et al., 1994; Nilsson et al., 1989), as well as mood and morale (Bonnet, 1994; Browne et al., 1994; Jacques et al., 1990). However, in the present study, performance on the SRT, a measure of new learning and memory, declined across two pre-sleep deprivation trials among all subjects, yet was not significantly affected by 36 hours of sleep deprivation.

Previous research has indicated that deficits in new learning and memory occur following sleep deprivation due to brief lapses in consciousness known as microsleeps. When sleep deprived, one may experience numerous, brief episodes of sleep during which it is impossible for one to attend to the presentation of new material. Further, microsleeps deny one the opportunity to

rehearse newly learned information so that it may be integrated into long-term memory (Bonnet, 1994; Johnson, 1982). Microsleeps occur most frequently during monotonous tasks of long duration, such as vigilance tasks where one is required to observe a computer screen for an extended period of time and identify target stimuli (Bonnet, 1994). Thus, it is possible that sleep deprivation failed to elicit cognitive deficits among sleep-deprived subjects due to the lack of an opportunity for lapses in consciousness, or microsleeps, to occur. The SRT and PASAT were relatively brief in duration (15 minutes and 20 minutes, respectively), and required continual contact with the experimenter and continual verbal responding on the part of the subject. Thus, subjects were afforded little if any time for microsleeps, as stimulus presentation was rapid, brief, and required an immediate verbal response. Although an attempt was made to maximize the fatigue level of subjects prior to the last testing session by a 50-minute administration of the DRI and DPT, it appears that the lengthy pre-test manipulation did not help to elicit cognitive deficits post-sleep deprivation. Further, the DRI and DPT may have been sufficiently engaging to promote central nervous system arousal, thereby helping to counteract the effects of sleep deprivation. Thus, in retrospect, it is likely that the experimental measures should have been at least 30-minutes in duration and the test trials sufficiently expanded to permit microsleeps to occur such that post-deprivation deficits in new learning and memory might have been observed (Donnell, 1969; Johnson, 1982): Neuropsychological tests such as the SRT and the PASAT are not designed to tap the effects of sleep

deprivation, and thus may insensitive to it. Such tasks might be modified for future research.

The detrimental effects of the relatively brief task parameters may have been compounded by the fact subjects knew sleep deprivation would end immediately upon completion of the final testing session. Haslam (1983) found that simply providing subjects with the knowledge that sleep deprivation would end in a few hours was sufficient incentive for subjects' performance to improve by 30-percent. Future research might thus present subjects with the false information that further deprivation and testing should be expected.

The continued improvement in performance noted for all groups across time on the PASAT must also be discussed. Gronwall (1977) reported that although significant practice effects have been noted between the first and second administration of the PASAT, practice effects between subsequent administrations are minimal. However, practice effects were noted between the second and third administrations of the PASAT in the present study. Continued practice effects may have counteracted the degradation in performance expected following sleep deprivation. It is important to note that practice effects between the second and third PASAT administration also may have been compounded by the motivational component of the test. As the PASAT is an extremely demanding measure of information processing, it is plausible that subjects find it challenging to attempt to improve their score across administrations. Highly motivating tasks have been found to show no significant performance decrements after one night of sleep loss (Johnson,

1982). Further, as subjects had ample free time during which to discuss this rigorous measure, inter-subject competition may further increased motivation to do well on the PASAT (Colquhoun, 1982).

Finally, the duration of sleep deprivation in the present study may not have been sufficiently long to disrupt performance on measures of information processing, new learning, and memory. Although previous studies have reported declines in attentional, psychomotor, and memory performance following 24 hours of sleep deprivation (Browne et al., 1994; Jaques et al., 1990; Lingenfelser et al., 1994), performance has been found to be adversely affected more easily and completely following 55 hours or more of sleep deprivation (Elkin & Murray, 1974; Donnell, 1969). A 36-hour sleep deprivation protocol may have been particularly brief for college students who consistently deprive themselves of sleep and function, cognitively, at a near optimal level (Hawkins & Shaw, 1992).

Summary and Implications

Results of the current investigation indicated that 36-hours of sleep deprivation did not result in deficits in information processing, new learning, or memory among mildly head injured subjects who have sufficiently recovered from their injury. It may be that, although subjects in the current study sustained a MHI, injury severity was relatively minor and resulted in minimal diffuse brain injury: The large majority of subjects in the current study were injured playing sports or after a fall, and therefore may not have been exposed to rotational forces of sufficient velocity to produce axonal shearing and

subsequent diffuse brain injury. In order for cognitive deficits to be observed following recovery from MHI, it may be necessary to examine only subjects with the greatest potential of having sustained diffuse brain injury. Thus, it may be beneficial to exclude those who were merely dazed following MHI and include only subjects whose injury rendered them briefly unconscious. Along those lines, obtaining a sample of subjects who sustained a MHI during a MVA may increase the level of head injury severity in the sample, as MVAs are more likely than other common sources of MHI to result in diffuse brain injury due to acceleration/deceleration forces associated with the accident.

The endorsement of PCS symptoms has been noted to covary with the experience of neuropsychological deficits following MHI. However, neither MHI nor uninjured subjects who endorsed symptoms of PCS at a subclinical level exhibited performance decrements on neuropsychological measures following sleep deprivation. It was originally proposed that subjects in the present study would exceed the score of MHI subjects without PCS, studied by Gouvier et al. (1992), by one and one-half standard deviations. Although subjects in the present study endorsed PCS symptoms at a level commensurate with MHI subjects without PCS, as measured by Gouvier et al., (1992), PCSC scores did not exceed those of Gouvier's (1992) MHI sample. Further, though subjects in the current study endorsed a level of PCS that was significantly greater than 50 percent of the population screened, the level of symptom endorsement was not great enough to fall in the significantly elevated range, and thus does not warrant the classification of PCS.

It may be that subjects were asymptomatic after their injury. Thus, the likelihood of experiencing injury-related symptoms at some later point would be significantly reduced. It is possible that subjects in the current sample reported experiencing symptoms of fatigue, headache, etc. listed on the PCSC due to current life stressors, and thus did not attribute these symptoms to a prior head injury. If this was the case, it is less likely that endorsement of PCS symptoms would covary with neuropsychological deficits, as reported in the head injury literature. In the future, it would be beneficial to assess the onset and duration of PCS symptoms and select only subjects who report experiencing symptoms of PCS immediately following MHI.

Finally, 36-hours of sleep deprivation did not adversely affect the cognitive performance of head injured subjects or uninjured controls. It may be that the period of sleep deprivation in the present study was not sufficiently long to disrupt performance on measures of information processing, new learning, and memory. Thirty-six hours of sleep deprivation may have been particularly brief for college students who have a fair amount of experience functioning, cognitively, under sleep deprived conditions.

The lack of an effect following sleep deprivation may have been due to the lack of opportunity for microsleeps to occur during the relatively intense and rapid performance demands of the SRT and PASAT. As attentional lapses most likely did not occur, subjects were able to attend to the presentation of stimuli, integrate information into long-term memory, and thus, perform at a level commensurate with pre-sleep deprivation performance. Further,

motivational effects that resulted from awareness that the sleep deprivation period was about to end may have kept cognitive performance from declining following sleep deprivation. One may be more likely to find performance decrements post-sleep deprivation if the measures used to assess performance are at least 30-minutes in duration, are monotonous, and do not require a verbal response.

In conclusion, the combined aspects of the minor nature of MHI experienced by subjects in the present study, the subclinical level of PCS symptoms reported by MHI and uninjured subjects, the relatively brief length of sleep deprivation, and lack of opportunity for episodes of microsleep to occur during testing may have precluded the hypothesized results from being obtained.

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APPENDIX A: INFORMED CONSENT

LOUISIANA STATE UNIVERSITY-BATON ROUGE CAMPUS
Consent Form

1. Study Title: The Effects of Sleep Deprivation on Information Processing, New Learning, and Memory in Mildly Head-Injured Subjects With Postconcussion Symptoms

2. Performance Sites: 218 Audubon Hall-Louisiana State University

3. Investigators:
Name: Brian Betz, M.A.
Michelle Plauche', M.A.
Department: Psychology
Telephone: (504)388-8745 - LSU
(504)383-6642 - Home

4. Study Purpose: Through participation, volunteers will help to better delineate the effects of sleep deprivation on cognition following mild head injury.

5. Subject Inclusion: The study will include volunteers aged 18 and over who meet criteria for having sustained one mild head injury.

6. Subject Exclusion: Subjects will be excluded for the following reasons: (1) if, following mild head injury, they were unconscious for greater than 20 minutes, were hospitalized for greater than 48 hours, or sustained radiologically measurable damage (e.g., skull fracture, subdural or epidural hematoma), (2) have a prior history of a neurological disorder, (3) currently meet criteria for a diagnosable psychiatric disorder.

7. Study Description: Subjects who meet inclusion criteria will be randomly assigned to either a sleep-deprived (SD) group or a non-sleep-deprived (NSD) group. All subjects will complete a measure of information processing and a measure of memory on three separate occasions over a 36 hour period (8 am day 1, 8 pm day 1, and 8 pm day 2). The NSD group will be released between testing sessions. The SD group will remain in the laboratory throughout the study.

8. Benefits: The study will not benefit subjects directly, but will help to further the understanding of the effects of sleep deprivation on cognition following mild head injury.

9. Risks: Fatigue is expected following sleep deprivation. As fatigue may adversely affect driving, volunteers will be asked to arrange for transportation home following the study. If a subject is unable to arrange for transportation, the investigator will arrange for transportation.

10. Alternatives: The study does not investigate additional hypotheses. Therefore, an alternative means of participation is not available.

11. Removal: Subjects will have fulfilled all study requirements when they have completed all three testing sessions (8 am day 1, 8 pm day 1, and 8 pm day 2).

12. Right to Refuse: Subjects may choose NOT to participate or withdraw from the study at any time. If a subject elects to withdraw from the study prior to completion, he/she will be awarded credit for hours of participation up to that point.

13. Privacy: The results of the study may be published. The privacy of participating subjects will be protected and the identity of subjects will not be revealed.

14. Release of Information: Subject information, including demographic data collected during pre-study screening and test data, will be reviewed by the principle investigators. If data is reviewed by additional investigators in the future, subject identity will be kept secret.

15. Financial Information: Subjects will not receive financial compensation. However, they will be awarded points that will allow them to earn extra credit in various undergraduate psychology courses. Further, food and beverages will be provided to subjects in the sleep-deprived group who are required to remain at the neuropsychology laboratory for the duration of the study.

16. Signatures:

The study has been discussed with me and all my questions have been answered. I understand that additional questions regarding the study should be directed to the investigators listed above. I understand that if I have questions about subject rights, or other concerns, I can contact the Vice Chancellor of the LSU Office of Research and Economic Development at 388-5833. I agree with the terms above and acknowledge I have been given a copy of the consent form.

_____

Signature of the Subject Volunteer Date

_____

Witness Date

_____

Investigator(s) Date

APPENDIX B: NEUROLOGICAL SCREENING

NEUROLOGICAL SCREENING

Name:_____ Gender: Male Female Age:_____
Handedness: Right Left Ambidextrous Year in College:_____
Social Security #:_____

Have you ever had:

1. a seizure: Yes No
2. a stroke Yes No
3. multiple sclerosis Yes No
4. encephalitis Yes No
5. meningitis Yes No
6. cancer Yes No
7. Are you currently taking any prescription medication? Yes No
   If yes, what?_____
8. May we contact you to participate in the additional stages of this study (which your instructor will describe) for additional credit? Yes No
   Telephone #_____

Have you ever had a head injury? Yes No
This includes being hit in the head and/or hitting your head, and waking up seconds or minutes later. This also includes hitting your head (or been hit in the head) and not been knocked unconscious, but feeling dazed, stunned, or disoriented afterward?

If you answered no to the question regarding head injury, you may stop here. If you answered yes, please complete the rest of this page.

1. How times have you hit your head (or been hit in the head) and been knocked unconscious, or felt stunned, dazed, disoriented, etc. afterward? _____
2. Approximately how long ago did your injury occur?_____
3. How were you injured? (Please circle one): car accident, fall, playing sports, fight/assault, other.
4. If knocked unconscious following a head injury, how long were you unconscious? (please circle one) 0 to 1 minute, 1 to 5 minutes, 5 to 10 minutes, 10 to 20 minutes, longer than 20 minutes.
5. Did you seek medical attention following your injury? Yes No
6. Were you hospitalized following your injury? Yes No
   a. If yes, how long did you remain in the hospital?_____
   b. Did you have a skull fracture, hematoma, or surgery because of your injury? Yes No. Did you have an MRI or CT scan? Yes No

APPENDIX C: POSTCONCUSSION SYNDROME CHECKLIST (PCSC)

APPENDIX B

POSTCONCUSSION SYNDROME CHECKLIST (PCSC)

NAME_____ DATE_____

Please rate the frequency, intensity and duration of each of the following symptoms based on how they have affected you over the past 2 months according to the following scale.

FREQUENCY	INTENSITY	DURATION
1 = Not at all	1 = Not at all	1 = Not at all
2 = Seldom	2 = Vaguely present	2 = A few seconds
3 = Often	3 = Clearly present	3 = A few minutes
4 = Very often	4 = Interfering	4 = A few hours
5 = All the time	5 = Crippling	5 = Constant

	FREQUENCY	INTENSITY	DURATION
Headache	_____	_____	_____
Dizziness	_____	_____	_____
Irritability	_____	_____	_____
Memory Problems	_____	_____	_____
Difficulty Concentrating	_____	_____	_____
Fatigue	_____	_____	_____
Visual Disturbances	_____	_____	_____
Aggravated by Noise	_____	_____	_____
Judgment Problems	_____	_____	_____
Anxiety	_____	_____	_____

Thank you for your time and effort in the completion of this form.

APPENDIX D: PEABODY PICTURE VOCABULARY TEST-REVISED (FORM L)

PPVT - FORM L

First Name:_____

Last 4 Digits of Social Security #:_____

110._____	132._____	153._____	175._____
111._____	132._____	154._____	
112._____	133._____	155._____	
113._____	134._____	156._____	
114._____	135._____	157._____	
115._____	136._____	158._____	
116._____	137._____	159._____	
117._____	138._____	160._____	
118._____	139._____	161._____	
119._____	140._____	162._____	
120._____	141._____	163._____	
121._____	142._____	164._____	
122._____	143._____	165._____	
123._____	144._____	166._____	
124._____	145._____	167._____	
125._____	146._____	168._____	
126._____	147._____	169._____	
127._____	148._____	170._____	
128._____	149._____	171._____	
129._____	150._____	172._____	
130._____	151._____	173._____	
131._____	152._____	174._____	

APPENDIX E: BECK DEPRESSION INVENTORY

INSTRUCTIONS TO THE BECK INVENTORY

On this questionnaire are groups of statements. Please read each group of statements carefully. Then pick out the one statement in each group which best describes the way you have been feeling the PAST WEEK, INCLUDING TODAY! Circle the number beside the statement you picked. If several statements in the group seem to apply equally well, circle each one. Be sure to read all the statements in each group before making your choice.

1. 0 I do not feel sad.
   1 I feel sad.
   2 I am sad all the time and can't snap out of it.
   3 I am so sad or unhappy that I can't stand it.

2. 0 I am not particularly discouraged about the future.
   1 I feel discouraged about the future.
   2 I feel I have nothing to look forward to.
   3 I feel that the future is hopeless and that things cannot improve.

3. 0 I do not feel like a failure.
   1 I feel I have failed more than the average person.
   2 As I look back on my life, all I see is a lot of failures.
   3 I feel I am a complete failure as a person.

4. 0 I get as much satisfaction out of things as I used to.
   1 I don't enjoy things the way I used to.
   2 I don't get real satisfaction out of anything anymore.
   3 I am dissatisfied or bored with everything.

5. 0 I don't feel particularly guilty.
   1 I feel guilty a good part of the time.
   2 I feel quite guilty most of the time.
   3 I feel guilty all of the time.

6. 0 I don't feel I am being punished.
   1 I feel I may be punished.
   2 I expect to be punished.
   3 I feel I am being punished.

7. 0 I don't feel disappointed in myself.
   1 I am disappointed in myself.
   2 I am disgusted with myself.
   3 I hate myself.

8. 0 I don't feel I am any worse than anybody else.
   1 I am critical of myself for my weaknesses or mistakes.
   2 I blame myself all the time for my faults.
   3 I blame myself for everything bad that happens.

9. 0 I don't have any thoughts of killing myself.
   1 I have thoughts of killing myself, but I would not carry them out.
   2 I would like to kill myself.
   3 I would kill myself if I had the chance.

10. 0 I don't cry any more than usual.
    1 I cry more now than I used to.
    2 I cry all the time now.
    3 I used to be able to cry, but now I can't cry even though I want to.

11. 0 I am no more irritated now than I ever am.
    1 I get annoyed or irritated more easily than I used to.
    2 I feel irritated all the time now.
    3 I don't get irritated at all by the things that used to irritate me.

12. 0 I have not lost interest in other people.
1 I am less interested in other people than I used to be.
2 I have lost most of my interest in other people.
3 I have lost all of my interest in other people.

13. 0 I make decisions about as well as I ever could.
1 I put off making decisions more than I used to.
2 I have greater difficulty in making decisions than before.
3 I can't make decisions at all anymore.

14. 0 I don't feel I look any worse than I used to.
1 I am worried that I am looking old or unattractive.
2 I feel that there are permanent changes in my appearance that make me look unattractive.
3 I believe that I look ugly.

15. 0 I can work about as well as before.
1 It takes an extra effort to get started at doing something.
2 I have to push myself very hard to do anything.
3 I can't do any work at all.

16. 0 I can sleep as well as usual.
1 I don't sleep as well as I used to.
2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.
3 I wake up several hours earlier than I used to and cannot get back to sleep.

17. 0 I don't get more tired than usual.
1 I get tired more easily than I used to.
2 I get tired from doing almost anything.
3 I am too tired to do anything.

18. 0 My appetite is no worse than usual.
1 My appetite is not as good as it used to be.
2 My appetite is much worse now.
3 I have no appetite at all anymore.

19. 0 I haven't lost much weight, if any, lately.
1 I have lost more than 5 pounds.
2 I have lost more than 10 pounds.
3 I have lost more than 15 pounds.

20. 0 I am no more worried about my health than usual.
1 I am worried about physical problems such as aches and pains; or upset stomach; or constipation.
2 I am very worried about physical problems and it's hard to think of much else.
3 I am so worried about my physical problems, that I cannot think about anything else.

21. 0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

APPENDIX F: STATE TRAIT ANXIETY INVENTORY

SELF-EVALUATION QUESTIONNAIRE

Developed by Charles D. Spielberger
in collaboration with
R. L. Gorsuch, R. Lushene, P. R. Vagg, and G. A. Jacobs

STAI Form Y-1

Name _____ Date _____ S ____

Age _____ Sex: M ____ F ____ T ____

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you feel *right* now, that is, *at this moment*. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

	NOT AT ALL	SOMEWHAT	MODERATELY SO	VERY MUCH SO
1. I feel calm	①	②	③	④
2. I feel secure	①	②	③	④
3. I am tense	①	②	③	④
4. I feel strained	①	②	③	④
5. I feel at ease	①	②	③	④
6. I feel upset	①	②	③	④
7. I am presently worrying over possible misfortunes	①	②	③	④
8. I feel satisfied	①	②	③	④
9. I feel frightened	①	②	③	④
10. I feel comfortable	①	②	③	④
11. I feel self-confident	①	②	③	④
12. I feel nervous	①	②	③	④
13. I am jittery	①	②	③	④
14. I feel indecisive	①	②	③	④
15. I am relaxed	①	②	③	④
16. I feel content	①	②	③	④
17. I am worried	①	②	③	④
18. I feel confused	①	②	③	④
19. I feel steady	①	②	③	④
20. I feel pleasant	①	②	③	④

Consulting Psychologists Press
577 College Avenue, Palo Alto, California 94306

SELF-EVALUATION QUESTIONNAIRE

STAI Form Y-2

Name _____ Date _____

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you *generally* feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

	ALMOST NEVER	SOMETIMES	OFTEN	ALMOST ALWAYS
21. I feel pleasant	①	②	③	④
22. I feel nervous and restless	①	②	③	④
23. I feel satisfied with myself	①	②	③	④
24. I wish I could be as happy as others seem to be	①	②	③	④
25. I feel like a failure	①	②	③	④
26. I feel rested	①	②	③	④
27. I am "calm, cool, and collected"	①	②	③	④
28. I feel that difficulties are piling up so that I cannot overcome them	①	②	③	④
29. I worry too much over something that really doesn't matter	①	②	③	④
30. I am happy	①	②	③	④
31. I have disturbing thoughts	①	②	③	④
32. I lack self-confidence	①	②	③	④
33. I feel secure	①	②	③	④
34. I make decisions easily	①	②	③	④
35. I feel inadequate	①	②	③	④
36. I am content	①	②	③	④
37. Some unimportant thought runs through my mind and bothers me	①	②	③	④
38. I take disappointments so keenly that I can't put them out of my mind	①	②	③	④
39. I am a steady person	①	②	③	④
40. I get in a state of tension or turmoil as I think over my recent concerns and interests	①	②	③	④

APPENDIX G: SELECTIVE EMINDING TEST: RECORD FORMS 2, 3, AND 4

SELECTIVE REMINDING TEST - Form 2

Name:_____ Age:_____

Circle: Day 1, 8am Day 1, 8pm Day 2, 8pm

	1	2	3	4	5	6	7	8	9	10	11	12
Shine												
Disagree												
Fat												
Wealthy												
Drunk												
Pin												
Grass												
Moon												
Prepare												
Prize												
Duck												
Leaf												

Recognition Trial

1. _____Shine	13._____Wake
2. _____Chime	14._____Lawn
3. _____Dispute	15._____Moon
4. _____Disagree	16._____Noon
5. _____Fat	17._____Husband
6. _____Trail	18._____Prepare
7. _____Wealthy	19._____Award
8. _____Stopwatch	20._____Prize
9. _____Blunt	21._____Duck
10._____Drunk	22._____Leaf
11._____Pin	23._____Bird
12._____Grass	24._____Leap

Total Recall: _____ (number recalled over 12 trials)
LTS: _____ (Words recalled twice in a row: sum over 12 trials).
30-minute Recall: _____ (maximum = 12)
30-minute Recog.: _____

SELECTIVE REMINDING TEST - Form 3

Name:_____ Age:_____

Circle: Day 1, 8am Day 1, 8pm Day 2, 8pm

	1	2	3	4	5	6	7	8	9	10	11	12
Throw												
Lily												
Film												
Discreet												
Loft												
Beef												
Street												
Helmet												
Snake												
Dug												
Pack												
Tin												

Recognition Trial

1. _____Throw	13._____Clue
2. _____Toss	14._____Road
3. _____Flower	15._____Helmet
4. _____Lily	16._____Bacon
5. _____Film	17._____Smoke
6. _____Slave	18._____Snake
7. _____Discreet	19._____Dog
8. _____Distinct	20._____Dug
9. _____Attic	21._____Pack
10._____Loft	22._____Tin
11._____Beef	23._____Bundle
12._____Street	24._____Shirt

Total Recall: _____ (number recalled over 12 trials)
LTS: _____ (Words recalled twice in a row: sum over 12 trials).
30-minute Recall: _____ (maximum = 12)
30-minute Recog.: _____

SELECTIVE REMINDING TEST - Form 4

Name:_____ Age:_____

Circle: Day 1, 8am Day 1, 8pm Day 2, 8pm

	1	2	3	4	5	6	7	8	9	10	11	12
Egg												
Runway												
Fort												
Toothache												
Drown												
Baby												
Lava												
Damp												
Pure												
Vote												
Strip												
Truth												

Recognition Trial

1. _____Egg	13._____Infant
2. _____Source	14._____Rock
3. _____Airline	15._____Damp
4. _____Runway	16._____Hook
5. _____Fort	17._____Purse
6. _____Sink	18._____Pure
7. _____Toothache	19._____Ballot
8. _____Headache	20._____Vote
9. _____Rib	21._____Strip
10._____Drown	22._____Truth
11._____Baby	23._____Chain
12._____Lava	24._____Fact

Total Recall: _____ (number recalled over 12 trials)
LTS: _____ (Words recalled twice in a row: sum over 12 trials).
30-minute Recall: _____ (maximum = 12)
30-minute Recog.: _____

APPENDIX H: PACED AUDITORY SERIAL ADDITION TEST RECORD FORM

PASAT
Record Form

Name: _____ Age: _____ Date: _____

2

7 (9)					8 (12)					5 (13)					
5 (12)					7 (15)					4 (9)					
1 (6)					1 (8)					8 (12)					
4 (5)					6 (7)					2 (10)					
9 (13)					3 (9)					1 (3)					
6 (15)					5 (8)					7 (8)					
5 (11)					9 (14)					5 (12)					
3 (8)					2 (11)					9 (14)					
8 (11)					7 (9)					1 (10)					
4 (12)					5 (12)					3 (4)					
3 (7)					3 (8)					6 (9)					
2 (5)					4 (7)					2 (8)					
6 (8)					7 (11)					9 (11)					
9 (15)					1 (8)					7 (16)					
3 (12)					5 (6)					8 (15)					
4 (7)					8 (13)					2 (10)					
5 (9)					3 (11)					4 (6)					
8 (13)					4 (7)					7 (11)					
6 (14)					6 (10)					6 (13)					
4 (10)					8 (14)					3 (9)					

	Total Correct	Time/Response
2.4 sec. pacing	_____	_____
2.0 sec. pacing	_____	_____
1.6 sec. pacing	_____	_____
1.2 sec. pacing	_____	_____
	Total time _____	Mean time _____

APPENDIX I: DRIVER PERFORMANCE TEST RECORD FORM

DRIVER PERFORMANCE TEST (DPT)

NAME(PRINT) _____ DATE_____ SCORE_____

CIRCLE THE MOST CORRECT ANSWER

P1. A B C D	5. A B C D	14. A B C D	23. A B C D	32. A B C D
P2. A B C D	6. A B C D	15. A B C D	24. A B C D	33. A B C D
P3. A B C D	7. A B C D	16. A B C D	25. A B C D	34. A B C D
P4. A B C D	8. A B C D	17. A B C D	26. A B C D	35. A B C D
P5. A B C D	9. A B C D	18. A B C D	27. A B C D	36. A B C D
1. A B C D	10. A B C D	19. A B C D	28. A B C D	37. A B C D
2. A B C D	11. A B C D	20. A B C D	29. A B C D	38. A B C D
3. A B C D	12. A B C D	21. A B C D	30. A B C D	39. A B C D
4. A B C D	13. A B C D	22. A B C D	31. A B C D	40. A B C D

****

TOTAL SCORE_____/ TOTAL S___ TOTAL I___ TOTAL P___ TOTAL D___ TOTAL E___

APPENDIX J: DRIVER RISK INDEX RECORD FORM

DRI RESPONSE FORM

NAME _____ DATE _____ DRI _____

EXAMPLE QUESTION: AGREE DISAGREE .

1. __	AGREE	DISAGREE	26. __	AGREE	DISAGREE
2. __	AGREE	DISAGREE	27. __	AGREE	DISAGREE
3. __	AGREE	DISAGREE	28. __	AGREE	DISAGREE
4. __	AGREE	DISAGREE	29. __	AGREE	DISAGREE
5. __	AGREE	DISAGREE	30. __	AGREE	DISAGREE
6. __	AGREE	DISAGREE	31. __	AGREE	DISAGREE
7. __	AGREE	DISAGREE	32. __	AGREE	DISAGREE
8. __	AGREE	DISAGREE	33. __	AGREE	DISAGREE
9. __	AGREE	DISAGREE	34. __	AGREE	DISAGREE
10. __	AGREE	DISAGREE	35. __	AGREE	DISAGREE
11. __	AGREE	DISAGREE	36. __	AGREE	DISAGREE
12. __	AGREE	DISAGREE	37. __	AGREE	DISAGREE
13. __	AGREE	DISAGREE	38. __	AGREE	DISAGREE
14. __	AGREE	DISAGREE	39. __	AGREE	DISAGREE
15. __	AGREE	DISAGREE	40. __	AGREE	DISAGREE
16. __	AGREE	DISAGREE	41. __	AGREE	DISAGREE
17. __	AGREE	DISAGREE	42. __	AGREE	DISAGREE
18. __	AGREE	DISAGREE	43. __	AGREE	DISAGREE
19. __	AGREE	DISAGREE	44. __	AGREE	DISAGREE
20. __	AGREE	DISAGREE	45. __	AGREE	DISAGREE
21. __	AGREE	DISAGREE	46. __	AGREE	DISAGREE
22. __	AGREE	DISAGREE	47. __	AGREE	DISAGREE
23. __	AGREE	DISAGREE	48. __	AGREE	DISAGREE
24. __	AGREE	DISAGREE	49. __	AGREE	DISAGREE
25. __	AGREE	DISAGREE	50. __	AGREE	DISAGREE

[illegible] TOTAL THE CIRCLE MARKS AND ENTER THE TOTAL NUMBER IN THE DRI BLANK AT THE TOP OF THIS FORM.

VITA

Brian Betz obtained a bachelor of arts degree from the California State University at San Diego. He obtained a master of arts degree and doctor of philosophy degree in clinical psychology at Louisiana State University. He completed his clinical internship with an emphasis in neuropsychology at Rush-Presbyterian-St. Luke's Medical Center in Chicago, Illinois. He is currently completing a post doctoral fellowship in geriatric neuropsychology at the University of California Los Angeles Neuropsychiatric Institute and Hospital.

DOCTORAL EXAMINATION AND DISSERTATION REPORT

Candidate: Brian Betz

Major Field: Psychology

Title of Dissertation: The Effects of Sleep Deprivation on Information Processing, New Learning, and Memory in Mildly Head-Injured Subjects with Postconcussion Symptoms

Approved:

Major Professor and Chairman

Dean of the Graduate School

EXAMINING COMMITTEE:

Date of Examination:

December 19, 1996